The New England
Journal of Medicine

Copyright, 1944, by the Massachusetts Medical Society

Volume 231 AUGUST 31, 1944 Number 9

CHRONIC LATENT HEPATITIS FOLLOWING CATARRHAL JAUNDICE*

MARK D. ALTSCHULE, M.D.,† AND D. ROURKE GILLIGAN, M.D.‡

BOSTON

THERE are now available in the literature records of the persistence of icterus,[1-8] as well as reports describing increased plasma bilirubin and decreased bilirubin excretory function[5-9] in some patients who years previously had had an attack of catarrhal jaundice. Chronic gastrointestinal complaints[5-9] and chronic hepatic enlargement[5-8] have also been reported as sequelae of acute catarrhal jaundice. The present report of laboratory and clinical studies made in an unselected series of 36 persons who had had catarrhal jaundice one to twenty-nine years previously, similarly demonstrates in some cases the persistence for years of certain evidences of hepatic dysfunction and affords information concerning the frequency of abnormal findings. This study was thought to be particularly pertinent at the present time because of the large number of cases of catarrhal jaundice in the armed forces of the present war; the incidence of the disorder in the War of 1812, the American Civil War, the Franco-Prussian War and World War I was similarly high. There is no complete accord in the interpretation of the clinical significance of so-called "chronic latent hepatitis" following catarrhal jaundice; the present study adds information bearing on this problem.

MATERIAL AND METHODS

The group studied was made up almost entirely of members of this hospital staff, students of the Harvard Medical School and of Harvard College and subjects who had been patients in this hospital at an earlier date when they had an attack of catarrhal jaundice. All these subjects had been observed during their episodes of icterus by competent physicians, including physicians on the staff of this hospital and in the Department of Hygiene of Harvard University. When actual hospital or physicians' records were not available, the symptoms and signs and the physician's diagnosis at the time of the attack were reviewed with the subject. The latter were, with one exception, scientists, technicians or medical students.

The bilirubin measurements were made by the method of Malloy and Evelyn, employing the Evelyn photoelectric colorimeter.[10] During the study the plasma bilirubin concentration was also measured in 36 normal persons with no history of hepatic disease and no signs of any visceral disorder. These subjects had plasma bilirubin levels ranging from 0.08 to 0.82 mg. per 100 cc., with an average of 0.31 mg., 32 subjects having values of 0.6 mg. or less (Fig. 1). These findings are essentially the same as those reported for normal subjects by Malloy and Evelyn,[10] using the same method, and by Kornberg.[8]

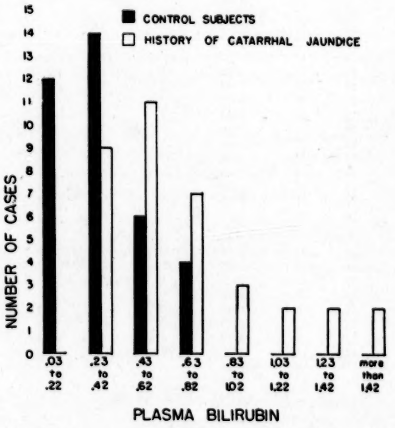

FIGURE 1. *Plasma Bilirubin Levels in Persons with and without a History of Catarrhal Jaundice.*

*From the Medical Service and the Medical Research Laboratories, Beth Israel Hospital, and the Department of Medicine, Harvard Medical School.

†Associate in medicine, Harvard Medical School; acting director, Medical Research Laboratory, Beth Israel Hospital.

‡Associate in medical research, Beth Israel Hospital; research associate, Harvard Medical School.

Observations

Plasma bilirubin levels. The plasma bilirubin concentrations ranged between 0.23 and 3.01 mg. per 100 cc. in the 36 patients who had recovered from infectious hepatitis; 16 had values above 0.60 mg. and 9, values above 0.82 mg. — that is, higher than any value found in the group of 36 normal subjects (Fig. 1). Four patients had had two or three attacks of jaundice. There was no relation between the concentration of plasma bilirubin and the occurrence of multiple attacks, nor was there any correlation of the former with the elapsed time between the attack and the present study (Table 1). The scleras appeared slightly icteric in 7 patients, all of whom had plasma bilirubin levels above 0.9 mg. per 100 cc. A yellowish tinge to the skin was present in 2 patients.

Hepatic and splenic enlargement. The liver was palpable 1 or 2 cm. below the costal margin in 9 patients, only 5 of whom had plasma bilirubin levels above the normal range. The spleen was felt in only 1 case, in which the plasma bilirubin concentration was 1.32 mg. per 100 cc.

Symptoms. Except for one patient with active peptic ulcer, another with active colitis and a third with a history of irritable colon many years previous to this study, none of the patients, icteric or not, had any chronic gastrointestinal complaints or any unusual number of gastrointestinal upsets. None showed lack of stamina; many of the subjects were or had been college athletes, including the one with the highest plasma bilirubin found, — namely, 3.01 mg. per 100 cc., — who was at the time of study a member of the Harvard crew. Two of the group, whose plasma bilirubin values were 0.89 and 1.98 mg., respectively, fifteen and twenty-one years after an attack of infectious hepatitis, had the skin changes of moderately severe vitamin A deficiency in spite of the ingestion of adequate diets.

Table 1. *Summary of Data.*

Case No.	Present Age	Date	First Attack: Icteric Index	First Attack: Palpable Liver	No. of Attacks	Period After First Attack	Present Observations: Plasma Bilirubin Level	Present Observations: Icterus of Scleras	Present Observations: Palpable Liver	Symptoms
	yr.					yr.	mg./100 cc.			
1	21	1939	?	+	3	3	3.01	+†	1 cm.	None
2	33	1920	?	?	1	21	1.97	+	1 cm.	Vitamin A deficiency
3	27	1936	90	1 cm.*	1	4 8/12	1.32	+	1 cm.‡	None
4	25	1926	?	?	1	15	1.24	+	0	None
5	18	1939	?	?	1	3 8/12	1.09	+	2 cm.	None
6	24	1932	?	?	1	10	1.09	+†	0	None§
7	20	1933	43	0	1	8	0.94	+	0	None
8	33	1928	?	?	1	15	0.89	0	0	Vitamin A deficiency
9	28	1932	?	0	1	9	0.85	0	0	None
10	35	1922	?	?	1	21	0.81	0	0	None
11	20	1941	?	?	1	2 6/12	0.81	0	0	None
12	29	1933	?	0	1	8	0.79	0	0	None
13	23	1938	100	?	1	2 7/12	0.79	0	0	None
14	22	1938	?	?	1	4	0.70	0	0	None
15	17	1937	50	0	1	3 7/12	0.63	0	2 cm.	None
16	33	1940	45	0	1	1	0.63	0	0	None
17	37	1917	?	?	2	25	0.61	0	0	Colitis for 3 yr.
18	20	1935	?	?	1	9	0.61	0	0	None
19	33	1913	?	?	3	29	0.54	0	0	None
20	35	1930	?	?	1	11	0.54	0	0	None
21	27	1922	?	?	1	20	0.54	0	0	None
22	27	1938	77	+	1	5	0.54	0	0	None
23	29	1940	60	3 cm.	1	1 1/12	0.47	0	2 cm.	None
24	25	1936	?	+	1	6	0.45	0	0	None
25	21	1939	?	?	1	2 10/12	0.43	0	2 cm.	None
26	25	1922	?	?	1	20	0.43	0	0	Irritable colon for several years after attack
27	32	1938	?	?	1	5	0.43	0	0	None
28	42	1940	?	0	1	1 9/12	0.38	0	0	None
29	47	1922	?	?	1	20	0.38	0	0	Peptic ulcer
30	22	1936	?	?	1	6	0.33	0	0	None
31	48	1930	100	+	1	12	0.33	0	0	None
32	22	1940	35	0	1	1 1/12	0.31	0	1 cm.	None
33	63	1934	?	?	1	8	0.29	0	1 cm.	None
34	22	1929	?	?	2	13	0.28	0	0	None
35	46	1928	?	?	1	15	0.25	0	0	None
36	57	1934	45	+	1	8	0.23	0	0	None

*Distance below costal margin.
†Skin also icteric.
‡Spleen also palpable.
§According to the patient's history, he had often had plasma bilirubin levels as high as 4.0 mg. per 100 cc.

Discussion

The disorder known as catarrhal jaundice has been shown to be a form of hepatitis[11, 12] apparently consequent to infection, although the causative agent has not been identified. It has been generally believed that almost all patients with catarrhal

jaundice recover completely. The occurrence of acute or subacute yellow atrophy followed by death or the development of clinical manifestations of chronic cirrhosis of the liver has been observed in occasional cases immediately after or shortly after an attack of infectious hepatitis; these immediate sequelae will not be discussed. That jaundice may be persistent in some patients following catarrhal jaundice has also been stated previously by a number of authors.[1–8] Studies of the plasma bilirubin concentration confirming this statement have been recorded.[5–8] In addition, impairment of bilirubin excretion after administration of bilirubin has been described by Soffer and Paulson,[9] Kalk,[5, 6] and Kornberg.[8] The results of the present study confirm these earlier findings. Our observation that abnormally high plasma bilirubin values persisted for years in 25 per cent of this series indicates a higher incidence of chronic mild impairment of hepatic function following catarrhal jaundice than has been generally believed to exist. The fact that impairment of bilirubin excretion is not infrequently demonstrated by the bilirubin-excretion test following catarrhal jaundice, even when there is no chronic hyperbilirubinemia,[8, 9] indicates that the incidence of chronic mild impairment of liver function is even higher than demonstrated by the 25 per cent incidence of chronic hyperbilirubinemia in our subjects. This concept is supported by the observation that the distribution of the plasma bilirubin values of the 27 patients who had had catarrhal jaundice but had bilirubin values within the normal range includes an abnormally large number of values in the high normal range (Fig. 1).

The significance of the persistently abnormal hepatic function after infectious hepatitis is not clear. A wide variety of vague chronic complaints, including dyspepsia, abdominal fullness, upper abdominal discomfort, anorexia, heartburn, fatigue and belching, are described in such patients by Kalk,[5, 6] Polack,[7] Kornberg[8] and, less emphatically, by Soffer and Paulson.[9] Kalk[6] also records complaints of a bad taste in the mouth, a feeling of tension, loss of ambition, headache, strong thirst, a craving for sweets and an aversion to alcohol and fat. In our own cases the icterus, when persistent, was not associated with chronic gastrointestinal complaints or a greater than normal incidence of acute gastrointestinal upsets; evidence of impaired utilization of vitamin A was, however, present in 2 cases. Enlargement of the liver, as well as that of the spleen in 1 case, was not associated with symptoms in these cases, although other authors[5–8] described hepatic enlargement in many of their patients with a variety of gastrointestinal complaints following catarrhal jaundice. It should be emphasized that our subjects were unselected. Thirty-four of them came to our attention as a result of our own efforts to secure subjects recovered from catarrhal jaundice and not because they sought medical advice. In 2 cases, the subjects required medical attention for symptoms unrelated to hepatic dysfunction.

The hepatic histopathologic changes responsible for the evidences of retention of bilirubin in these patients are unknown. The prognosis also is not clear. The occasional occurrence of cirrhosis of the liver years after the apparent complete recovery from what appears to have been catarrhal jaundice has been noted by some clinicians,[13] but, this must be an extremely infrequent occurrence if one is to judge from the relative frequencies of catarrhal jaundice and of hepatic cirrhosis.

It is concluded, therefore, that the chronic latent hepatitis that often follows apparent recovery from catarrhal jaundice is usually benign and unassociated with symptoms and is therefore not clinically significant; in rare cases it is the forerunner of the clinical syndrome cirrhosis of the liver.

Summary and Conclusions

Studies of 36 persons who had had attacks of catarrhal jaundice one to twenty-nine years previously revealed hyperbilirubinemia in 25 per cent. It is probable that the incidence of impairment of liver function was even higher, since the distribution of plasma bilirubin values revealed an abnormally large number in the high normal range. Nine subjects in the entire group had palpable livers and 1 a palpable spleen. The chronic latent liver disorder revealed by this study was not accompanied by symptoms and did not appear to be progressive. It is concluded that a mild, benign form of chronic hepatitis is frequent after catarrhal jaundice.

We are indebted to Drs. Arlie V. Bock and Andrew W. Contratto, of the Hygiene Department, Harvard University, and Drs. Lewis Dexter and Lewis W. Kane, of the Harvard Medical School, for their help in obtaining many of the subjects of this study.

References

1. Meakins, J. C. *The Practice of Medicine*. 1430 pp. St. Louis: The C. V. Mosby Company, 1940.
2. Christian, H. A. *The Principles and Practice of Medicine: Designed for use of practitioners and students of medicine*. Fourteenth edition. 1475 pp. New York: D. Appleton-Century Company, 1942.
3. Bloomfield, A. L. Diseases of the alimentary tract. A section in *Internal Medicine: Its theory and practice*. Edited by John H. Musser. Third edition. 1438 pp. Philadelphia: Lea and Febiger, 1938. P. 715.
4. Johnson, T. A., and Bockus, H. L. Significance of mild hyperbilirubinemia in gastrointestinal patients. *J. A. M. A.* **121**:729-733, 1943.
5. Kalk, H. Klinische Untersuchungen über die Frage des latenten Leberschadens. I. Untersuchungen mit der Bilirubinbelastungsprobe beim Ikterus catarrhalis. *Deutsche med. Wchnschr.* **58**: 1078-1080, 1932.
6. *Idem*. Klinische Untersuchungen über die Frage des latenten Leberschadens. II. Klinische Untersuchungen bei den sogenannten Hepatopathien nach überstandenem Ikterus catarrhalis. *Deutsche med. Wchnschr.* **58**:1119-1122, 1932.
7. Polack, E. Chronic hepatitis in young persons, with or without intermittent jaundice. *Acta med. Scandinav.* **93**:614-621, 1938.
8. Kornberg, A. Latent liver disease in persons recovered from catarrhal jaundice and in otherwise normal medical students as revealed by bilirubin excretion test. *J. Clin. Investigation* **21**:299-308, 1942.
9. Soffer, L. J., and Paulson, M. Residual hepatic damage in catarrhal jaundice as determined by the bilirubin excretion test. *Arch. Int. Med.* **53**:809-813, 1934.
10. Malloy, H. T., and Evelyn, K. A. Determination of bilirubin with the photoelectric colorimeter. *J. Biol. Chem.* **119**:481-490, 1937.
11. Roholm, K., and Iversen, P. Changes in liver in acute epidemic hepatitis (catarrhal jaundice) based on thirty-eight aspiration biopsies. *Acta path. et microbiol. Scandinav.* **16**:427-442, 1939.
12. Dible, J. H., McMichael, J., and Sherlock, S. P. V. Pathology acute hepatitis: aspiration biopsy studies of epidemic, arsenotherapy and serum jaundice. *Lancet* **2**:402-408, 1943.
13. Ratnoff, O. D., and Patek, A. J., Jr. Natural history of Laennec's cirrhosis of liver: analysis of 386 cases. *Medicine* **21**:207-268, 1942.

DISLOCATION OF THE KNEE JOINT*

A Report of Two Cases

JAMES WARREN SEVER, M.D.†

BOSTON

THIS is a report of 2 cases of dislocation of the knee joint — one an anterior dislocation of the knee and the other a compound posterior medial dislocation.

CASE 1. The patient, a 47-year-old carpenter, was admitted to the Cambridge Hospital on June 29, 1942, shortly after falling 15 feet from a ladder. His right leg was in a Thomas splint, which had been applied by the police. Inspection showed an anterior dislocation of the knee (Fig. 1), with the distal end of the femur causing great pressure on the skin in the popliteal space. An anesthetic was immediately given, and the dislocation was reduced on a Bell table without great difficulty. In effecting the reduction a heavy canvas strap was placed around the back of the thigh just above the knee, and traction was applied on the lower leg in the line of deformity, with slight flexion at the same time. Following this, a plaster spica was applied from the toes, with the knee flexed at an angle of 20 or 25°. A check-up x-ray film showed no evidence of fracture and complete reduction. Before the dislocation was reduced, there was some obvious interference with the circulation in the toes, but after reduction the circulation was completely restored. The patient wore the spica from June 29 to July 13, when a new cast was applied from the toes to the groin. At that time there was no swelling of the knee joint, the skin was in good condition, and circulation was satisfactory.

On July 29, the cast was bivalved and the leg was inspected. There was good muscle strength, no swelling of the knee and some active motion in the knee joint. There was no apparent lack of stability, but there was still some evidence of ecchymosis about the knee.

The patient was discharged on August 2, with the leg still in the bivalved cast, which was removed on September 1. Following this he began to bear weight on the leg, first with crutches and then with a cane, and got about very well. During this period he received physiotherapy for the development of the quadriceps muscle. On September 15, it was noted that he was walking with a marked toe drop and had considerable weakness in the peroneal and tibial groups, as well as in the dorsal flexors of the foot. As a result of this condition, a short caliper splint was applied to prevent toe drop, with marked improvement in walking. The patient continued his physiotherapy. By October he had made considerable improvement, and in November the strength of the dorsal flexors, tibials and peroneals had increased almost to normal. He was still wearing his brace, but in the latter part of November he had improved to such an extent that it was removed. At that time he had excellent strength in the quadriceps and had returned to his work. When last seen on February 5, 1943, the knee showed no evidence of pain, discomfort or instability. There was no evidence of any injury to the semilunar cartilages, nor was there evidence of instability due to injury of the crucial ligaments, that is, undue mobility of the tibia on the femur with the leg extended or the knee flexed. The knee could be fully extended and could be flexed 10° beyond a right angle, and the joint was in excellent weight-bearing position. Full power in the dorsal flexors and other muscles of the lower leg had been recovered, and there was no atrophy.

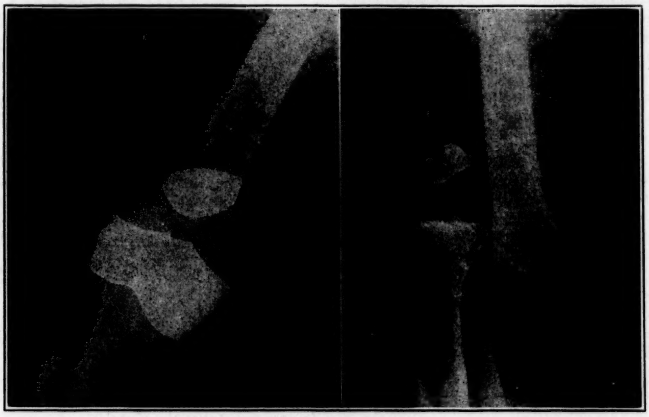

FIGURE 1. *Roentgenograms Showing an Anterior Dislocation of the Right Knee (Case 1).*

CASE 2.‡ The patient, a 39-year-old telegraph operator, was struck from behind by an automobile on March 14, 1942, suffering a posterior medial dislocation of the left knee (Fig. 2), compounded into the popliteal space through a 7-cm. wound, with periosteal stripping of the lower end of the femur (Fig. 2), as well as a compound comminuted fracture of the upper third of both bones of the lower right leg. He

*From the Orthopedic Clinic, Cambridge Hospital, Cambridge, Massachusetts. Read at a meeting of the Boston Surgical Society, February 7, 1944.

†Assistant professor of orthopedic surgery, Harvard Medical School; orthopedic surgeon, Cambridge Hospital and Children's Hospital.

‡I am indebted to Dr. G. Guy Bailey, Jr., for permission to report this case.

was treated for 8 weeks in a plaster cast after reduction of the dislocation and repair of the compound wound, and so far as the dislocation was concerned made a perfect recovery. It took considerably longer to repair the condition of the other leg. It was 4 weeks after removal of the cast before the patient attempted weight bearing on the left leg. With physiotherapy, he made a perfect recovery so far as the dislocation was concerned, and eventually the comminuted fracture of the other leg healed, so that he was able to resume his work.

At examination on February 12, 1944, the left leg was straight, with excellent muscle strength and full extension at the knee joint, which could be flexed 10° beyond a right angle. There was slight lateral instability or increased motion in the joint. There was no evidence of any injury to the semilunar cartilages as manifested by swelling, locking or instability, and no pain or discomfort. Furthermore, there was no evidence of instability due to injury to the crucial ligaments. In spite of the extensive injury, no injury to the nerves or vessels had been sustained.

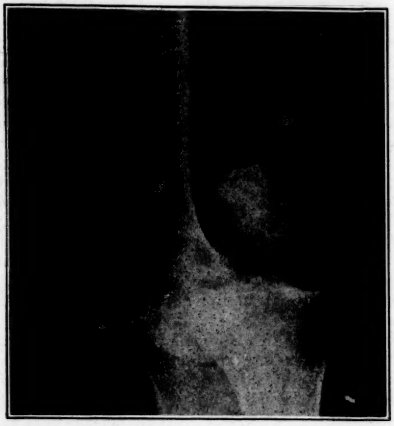

FIGURE 2. *Roentgenogram Showing a Posteromedial Dislocation of the Left Knee (Case 2).*

DISCUSSION

In the literature on dislocations of the knee,[1-7] it is emphasized by the various authors that there is total destruction of various ligaments in and about the knee joint. Injuries to the nerves, particularly the peroneal nerve,[1] due to stretching, is not infrequent. In one case the sciatic nerve was completely severed. Occasional cases of thrombosis of the posttibial as well as the popliteal artery have been reported,[1, 2] these cases requiring amputation.[3] The nerve injury, except for the cases showing complete section, usually healed, as was true in Case 1 of this report.

In discussing dislocations of the knee joints, one speaks in terms of displacement of the lower leg; that is, an anterior dislocation means a forward displacement of the tibia on the femur and so forth. This condition is extremely rare, and Ritter[4] reports that in 23,000 accident cases in the Reconstruction Hospital in New York, dislocation of the knee joint occurred in only 1 case, and that this was not a complete dislocation. He adds that at the New York Post-Graduate Hospital there were only 3 cases in a long period of years. All the authors speak of the comparative ease of reduction, which, of course, is to be expected considering that all the structures about the knee joint have been torn. In one or two reports open operations have been done to reduce the dislocations, but this does not seem indicated.[5] Most authors believe that fixation for six or eight weeks, later combined with physiotherapy and sometimes with a knee cage splint, is a better form of treatment. Others believe that passive motions begin in ten days, with early weight bearing at fourteen days, is the proper method. It seems wiser to me to wait until the structures about the knee joint have undergone repair before abusing them by early motion and weight bearing.

The most frequent type of dislocation about the knee is the anterior one, which occurred in 40 per cent of the cases reported. This takes place when the knee is in full extension. Posterior dislocations occurred in 20 per cent and result when the knee is in flexion. Lateral dislocations occurred in 20 per cent, and medial ones in 7 per cent. Which of the two latter occurs of course depends on whether the force is applied from the inner or the outer side of the leg. It is interesting to note that in many cases, even with such a severe injury, a satisfactory, stable and excellently functioning knee joint has resulted.

SUMMARY

Two cases of dislocation of the knee joint are reported, and the pertinent literature is discussed.

321 Dartmouth Street

REFERENCES

1. Ferguson, J. A., and Allen, L. Complete medial dislocation of knee joint with division of common peroneal nerve. *J. Bone & Joint Surg.* **21**:1012-1014, 1939.
2. Conwell, H. E., and Alldredge, R. H. Dislocations of knee joint, with report of complete external lateral dislocation. *J. A. M. A.* **106**:1252-1256, 1936.
3. Anderson, R. L. Dislocation of knee: report of four cases. *Arch. Surg.* **46**:598-603, 1943.
4. Ritter, H. H. Dislocation of knee joint with report of case. *J. Bone & Joint Surg.* **14**:391-394, 1932.
5. Fichman, A. M. Complete lateral dislocation of knee joint. *J. A. M. A.* **105**:1111-1113, 1935.
6. Mitchell, J. I. Dislocation of knee; report of four cases. *J. Bone & Joint Surg.* **12**:640-646, 1930.
7. Lothrop, H. A. Forward dislocation of knee. *Boston M. & S. J.* **158**: 324, 1908.

BOSTON MEDICAL LIBRARY

Report of the President*

IT IS the privilege of our librarian, Dr. Viets, to give an account of our professional activities, of the state of our collections of books, periodicals and memorabilia and of our services to the medical and lay public. The duty of the president is to make known to the fellows and friends of the Library its physical and financial condition, its needs, its projects and its hopes.

The year 1943, the second of our country's complete preoccupation with a desperate global war, has naturally found the Library striving to maintain its facilities and functions rather than to extend its undertakings in a way that would necessarily be incompatible with the war effort. We have not been hibernating, but our circulation and metabolism have suffered inevitable reduction, from which, when peace reigns again, we shall rally to renewed activity and wider usefulness.

There has been a net gain in our membership of twenty-eight. During the year eight fellows have died, as follows: Dr. Seth F. Arnold, Dr. Charles S. Butler, Dr. C. Macfie Campbell, Dr. Isador H. Coriat, Dr. Charles C. Foster, Dr. Amos I. Hadley, Dr. George A. Leland and Dr. Halbert G. Stetson. The loyalty of many of these men who, though they had retired and probably no longer needed the Library, continued actively to support it merits a word of appreciation. Dr. Foster, for instance, did not let his eighty-seven years serve as a reason for resigning; perhaps he continued to enjoy browsing in the stacks or passing a comfortable hour of reading in the Prince Room. We are proud to record that one hundred and fifty-nine of our members are in active service and that we have done what we could to acknowledge our debt to them by remitting their dues.

In my last annual report was recorded the decision of the Rationing Board to reduce our supply of fuel oil to a point incompatible with a continuance of our activities as a going concern, and our consequent contract with the Boston Edison Company to make suitable installations and supply steam for heating. This conversion was promptly and skillfully made, and the new system has been adequate and satisfactory, but it has resulted in an increase of operating expense due not only to the increased cost of service but also to the loss of $1100 that we received annually by supplying heat under the old system to the Massachusetts Historical Society. We are grateful, however, that we are warmed and sheltered, and able to pursue our work with no more serious interruptions than practice air-raid alarms.

The financial statement and balance sheet have been examined and approved by highly responsible certified public accountants. Last year our income from fellowship and membership dues was $497 less than the year before; this year there has been a further decrease of $411. Our income from investments, however, shows a gratifying increase of $624, and our actual operating balance sheet shows a deficit considerably less than the $1305 recorded a year ago. On the other hand, we have had to charge against capital approximately $9500 to meet the cost of the conversion to steam heat and of structural alterations and renovations to improve the offices of the Massachusetts Medical Society and its organ, the *New England Journal of Medicine*, and of the *Journal of Bone and Joint Surgery*, which is the official publication of the American Orthopaedic Association. This money has been found in part by a loan secured from a commercial bank and in part by borrowing from the unexpended balance of our own restricted funds — a debt that of course must be repaid with the same fidelity as the bank loan. And mention of our financial debts cannot be dismissed without a reminder that our loan from the Massachusetts Medical Society, while somewhat reduced, still stands at $19,000.

The corporation and friends of the Boston Medical Library must look more deeply into our affairs than merely to scrutinize the balance sheet of our current operations. Actuated by principles of honorable frugality, the trustees have made both ends meet by the most scrupulously careful budgeting. But the Library is in danger of stagnation unless its resources can be materially increased. Our house is in good condition, but it is unfinished. There is room for five additional tiers of steel stacks that were provided for in the original plans, and lacking which our accessions of books and periodicals are piled in the cellar and in passageways. The periodical room is a barnlike hall with the roughest sort of filing alcoves and furniture. Holmes Hall needs refitting and rearrangement to make it really adequate as a reading and delivery room. But more than anything we need improved service. Our director, Mr. Ballard, although he has done better than Ponce de León in his search for the mythical fountain, cannot be expected to carry his heavy burden indefinitely, and a young man of promise should be working by his side, absorbing his familiarity with the Library and its treasures and his skill in library administration — the fruit of fifty years' experience. Our research librarians and cataloguers should likewise have apprentices. A sum of $100,000 would take care of the improvements in plant, and an additional income of $10,000 per year would enable us to lead with authority in the plans already made to correlate and unite the various working collections of medical books in and around Boston in a truly co-operative and mutually helpful way. We

*Read at the annual meeting, March 7, 1944.

must search for and inspire generous donors to help us with gifts and to remember us in their wills. In spite of the decision of the trustees of a charitable trust that we recently approached that they must deny our request because we obviously had a constituency to whom we could look for support, we must convince public-spirited citizens that the earnings of physicians are rarely sufficient to enable them to make capital contributions.

There are cheerful things to record, as well as needs. The trustees were touched to be told by the executors of the will of the late Annie G. Farlow, widow of our former librarian and enthusiastic supporter, Dr. John W. Farlow, that she had made an unrestricted bequest to the Library of $10,000 in memory of her husband. Dr. Farlow's avocation was the Library, and to it he gave without stint services more valuable than money. At his death heb equeathed $10,000, which was applied to urgent needs. His widow paid for the installation of the elevator and has helped us in other ways, and now posthumously adds another to our reasons for gratitude. The Massachusetts Medical Society and the *Journal of Bone and Joint Surgery* have confirmed their alliance with us and have increased their contribution to our support in return for the enlarged space and improved facilities that we have been able to afford them. The Suffolk District Medical Society has maintained its generous contribution, adding to the sum specifically due us for entertaining them. The James F. Ballard Publication Fund, which we started last year, has financed the publication of the *Catalogue of the Medieval and Renaissance Manuscripts and Incunabula in the Boston Medical Library*, which has been received with great approval by medical librarians and bibliophiles. It is our hope that this fund may receive constant additions, so that publications based on its treasures may be made by the Library from time to time.

As president I cannot close this brief report without paying tribute to the faithful service rendered by those responsible for the well-being of the Library. The trustees have attended the regular meetings of the Board and of standing committees in spite of exacting duties elsewhere. Of the executive officers, who receive no compensation, the secretary, the treasurer and the librarian must give much time to their work, especially the last, whose activity governs and regulates our whole endocrine system. Our director, Mr. Ballard, and the faithful group of research librarians and cataloguers continue the traditions of many years. They deserve the help and support of the corporation of the Boston Medical Library and of a wider public.

DAVID CHEEVER, *President*

MEDICAL PROGRESS

THE LATE EFFECTS OF TOTAL AND SUBTOTAL GASTRECTOMY*

FRANZ J. INGELFINGER, M.D.†

BOSTON

RESECTION of the human stomach, particularly of the distal half, has become a practical surgical procedure that carries a remarkably low mortality rate. In some clinics, gastric resections for malignant tumors are attended by a mortality rate of less than 10 per cent, whereas those carried out for benign lesions have a mortality risk of only 1 to 3 per cent.[1–6] It is therefore not surprising that gastrectomy is being performed with ever-increasing frequency, not only for gastric neoplasms but also in the treatment of peptic ulcers of the stomach, duodenum and jejunum.

In the case of cancer, gastric resection may have to be most extensive, sometimes total. In the case of ulcer, the amount of stomach removed varies with the operator and the particular problem presented by the patient, but many surgeons recommend that the gastric antrum and at least two thirds of the stomach be removed, under the reasonable conviction that the chances of a recurrent ulcer are decreased by leaving a gastric remnant that has little or no capacity to form free hydrochloric acid.[3, 6–9] In short, one purpose in undertaking a gastrectomy for ulcer is the suppression of the normal secretory functions of the stomach.

Can man live without a stomach or with a stomach whose functions are grossly impaired? The answer to this question has always been "Yes," but from time to time it is reported that life under such conditions is not invariably normal. In this review, recent evidence bearing on this problem is considered, for the present popularity of gastrectomy warrants an appraisal of any possible late effects that may follow total or subtotal removal of the stomach. The immediate postoperative complications are not discussed, nor does this review deal with the undoubted efficacy of gastrectomy in the treatment of gastric neoplasms and of

*From the Evans Memorial, Massachusetts Memorial Hospitals, and the Department of Medicine, Boston University School of Medicine.

†Assistant professor of medicine, Boston University School of Medicine.

many peptic ulcers. So far as possible, only those effects that can be directly ascribed to resection of gastric tissue are reviewed, but it is probable that some of the symptoms for which gastrectomy has been blamed have actually been caused by postoperative ulcer, gastritis or jejunitis. Allowance must also be made for the fact that many of the data presented by various authors have not had the benefit of a careful statistical analysis, as has been recommended by Hollander and Mage.[10]

Clinical Observations

Among those patients who have undergone gastrectomy, a clear majority find their mode of life unaffected by the fact that half or more of the stomach has been removed. On the other hand, many patients find it difficult to gain weight, some suffer from postcibal distress, and a few are subject to diarrhea. In a series of 104 patients with subtotal gastric resection, Church and Hinton[11] found that 47 per cent lost, 39 per cent gained and the remainder maintained their weight in a postoperative period that averaged nearly three years. Somewhat similar is the experience of Browne and McHardy,[12] who followed 30 gastrectomized patients for periods ranging from two to eleven years after operation and noted that 20 of these lost weight. Miller,[5] reporting a series of 173 cases, states that the majority did not gain weight easily, but he records actual weight loss in less than 10 per cent. This figure is more in line with the results reported in earlier series.[13–15]

The value of statistical analysis is exemplified by a paper by Santy and Mallet-Guy.[16] These authors divided their material according to the length of the postoperative period and showed that whereas 50 per cent of the patients followed for one to three years failed to regain their normal weight, only a few of the patients observed over longer intervals experienced difficulty in putting on weight. Furthermore, of the 50 per cent who failed to regain their normal weight, only one third — that is, 17 per cent of the whole group — failed to maintain their preoperative weight.

Total gastrectomy in young animals arrests growth and weight gain,[17, 18] and even adult animals without stomachs may lose weight[19] or require a higher dietary intake than does the normal animal.[20] In man, Farris, Ransom and Coller,[21] and Joll and Adler[22] indicate that maintenance of weight does not present a serious problem after total gastrectomy, but Rekers, Pack and Rhoads[23] observe that patients seldom return to their original weights after total removal of the stomach. It is interesting that appetite may be unimpaired after complete gastrectomy.[22, 24]

The postcibal distress experienced by some gastrectomized patients follows a fairly typical pattern. Even while the patient is still at his meal, and although his appetite may not be assuaged, a marked sensation of epigastric pressure, distention and fullness develops that prevents him from taking another morsel. Nausea may accompany these symptoms, and occasionally weakness, dizziness, perspiration and palpitation occur within an hour after the meal. To relieve his distress, the patient may belch, induce vomiting or assume certain positions that he has discovered to be effective in mitigating his symptoms. The incidence of a persistent postcibal symptom complex following subtotal or total gastrectomy is probably about 10 per cent,[11, 13–15] although reports on this point vary. A rather dark picture is drawn by Jordan,[25] who found that abdominal symptoms occurred postoperatively in 13 of 20 patients operated on for severe duodenal ulcer, in 18 of 41 operated on for bleeding ulcer and in 8 of 12 operated on for stomal ulcer. This series, however, includes ulcer recurrences and other complications. In discussing Jordan's paper, Mateer[26] states that after subtotal gastrectomy 14 per cent of his patients suffered from mild and 4 per cent from severe symptoms that could not be ascribed to ulcer recurrences. Of Browne and McHardy's[12] 30 patients, 16 complained of dyspepsia. On the other hand, only 4 of the 74 patients followed by Vitkin[27] had epigastric complaints. Three fourths of 113 patients observed by Weissenborn[28] were absolutely free of symptoms, about 20 per cent had mild distress, and only 5 per cent had symptoms that interfered with their daily routines. In general, failure to gain weight is particularly noticeable in the group of patients complaining of gastrointestinal symptoms, but the converse does not hold; many underweight patients are completely symptom free.

Frank diarrhea with cramps has been reported as a sequel to gastrectomy,[12, 29] but it is not very frequent. Many patients, however, find that their stools are soft or even mushy following the operation.

It is emphasized by many writers that the symptoms that can be ascribed to total or subtotal gastrectomy tend to decrease in frequency and intensity as the interval between the operation and the follow-up observations lengthens. Much of the distress present during the first postoperative year tends to disappear subsequently. This improvement is brought about partly by the patient's realization that he must avoid large meals and that he can get along comfortably on six small meals a day. An equally important cause for improvement, however, is the capacity of the gastrointestinal tract to adjust itself to new conditions.

Gastrointestinal Motility

After subtotal gastrectomy, the normal motor functions of the stomach and upper intestines are deranged. Fluoroscopic observations reveal that in some cases material leaves the gastric stump with difficulty, but much more frequently the con-

trast medium enters the intestine with such rapidity that the term "dumping stomach" has been used under these conditions. In an attempt to lessen the subsequent motor disorders, many modifications of the method of gastric resection have been proposed, and a considerable argument has developed in the European literature concerning the relative advantages of the Billroth I and Billroth II operations. No conclusive evidence, however, has as yet been presented that one type of operation alters normal gastrointestinal motility less than does another.[27] As will be indicated later, the gastrointestinal tract makes functional adjustments irrespective of the initial surgery, and other factors besides the exact size and position of the stoma influence the rate of gastric evacuation.

In the average patient who makes a good recovery from a subtotal gastrectomy, roentgenologic examination of the gastric remnant shows that this may be somewhat atonic in the early postoperative period, but subsequently it becomes small, appears to exhibit a high degree of tone and is often without a gas bubble.[14, 16, 27] Ingested barium enters the small intestine rapidly, so that the gastric emptying time is usually much less than that of the normal stomach.[15, 30] In time, however, the capacity of the gastric stump increases, a process that is effected by elongation of the greater curvature, the gas bubble reappears, and the stomach again assumes some of its reservoir functions. The partially resected stomach sometimes exhibits peristaltic waves,[27, 31] but in other cases peristalsis is not seen,[32] and the general motility pattern tends to be abnormal.[15]

Eventually, gastric emptying may occur intermittently and rhythmically, a phenomenon that has suggested to some that a new sphincter is formed by the gastric muscle that adjoins the stoma.[14, 30] Schindler[33] states that the stoma, as seen through the gastroscope, usually appears patent and rigid, but that in 4 out of 41 patients who had undergone resection for ulcer a rhythmic, pyloruslike activity of the sphincter was seen. Most observers do not believe that a new sphincter forms at the site of the stoma, Vitkin[27] maintaining that the stomal aperture is opened and closed by the efferent jejunal loop.

Immediately distal to the anastomosis, the small bowel may dilate and form small accessory reservoirs in which the intestinal content pools before being transported farther down the intestine. Particularly if the gastric remnant is small or if total gastrectomy has been performed, jejunal reservoirs may become large and permanent.[23] Beyond these dilated areas, motility of the small intestine is said to be essentially normal,[21, 23] or at least the contrast meal reaches the cecum and leaves the small bowel at a normal rate. In the growing monkey, however, puddling and dilatation occur in the lower small intestine after total gastrectomy.[18]

Unfortunately, the adjustments of the alimentary canal to subtotal gastrectomy do not proceed smoothly in all patients. Irregular or delayed emptying of the gastric stump may be marked, particularly in the early postoperative period. When emptying is delayed, the usual tendency is to focus all attention on the stoma in an effort to ascertain whether it is too narrow or is improperly placed, or whether there is some mechanical defect in its proximity.[34] The fact that many a so-called "nonfunctioning" stoma appears patent when the patient is reoperated on presented somewhat of a riddle until Ravdin and his associates[35, 36] showed that hypoproteinemia may retard gastric evacuation, both through the pylorus or through a gastroenteric anastomosis. Since then the explanation of hypoproteinemia has been readily invoked to account for many postoperative disorders of gastrointestinal motility, although the evidence for hypoproteinemia may be quite meager. Hypoproteinemia undoubtedly can exert a marked influence on motility,[37, 38] but other factors are at play, as indicated by the studies of Chauncey and Gray,[39] who could not correlate the level of the serum proteins and the rate of gastric emptying through a stoma.

The overemphasis that has been placed on the stoma in explaining abnormal gastrointestinal motility after gastrectomy can be ascribed to the concepts that have been held concerning the emptying mechanism of the normal stomach. In these concepts, the role of the pylorus has occupied a dominant position. Quigley,[40] however, has shown that in dogs the role of the pylorus is secondary and that gastric evacuation is chiefly controlled by the gastroduodenal pressure gradients. A few observations obtained by Abbott and his collaborators[41] suggest that the same conditions obtain in man. It follows that poor gastric evacuation after subtotal gastrectomy in some cases has nothing to do with the stoma but is produced by a flattened or reversed gastroenteric pressure gradient.

The effect of certain foods on the motility of the upper gastrointestinal tract illustrates the role played by pressure gradients. When fats, hydrochloric acid or hypertonic solutions enter the intestinal loops distal to the anastomosis, the resected as well as the normal stomach exhibits delayed emptying.[30, 42] This delay, it is now known, is produced by relaxation of the gastric musculature, not by sphincteric contraction. The consequent fall in intragastric pressure may be accompanied, in the case of gastrectomy, by a rise in intraenteric pressure, probably brought about by one of two mechanisms. Either the sudden influx of food activates the motor activity of the small intestine,[32, 42] or the volume of the intestinal contents is rapidly increased by an outpouring of enteric juices, which take over the diluting functions normally carried out in the stomach.[43] The net result of these pres-

sure changes is a flattening of the gastroduodenal or gastrojejunal gradients, with delayed gastric evacuation. To prove that abnormal gradients account for some of the disorders usually ascribed to a defective stoma, better devices for recording intraenteric pressures are needed, but Ravdin, Royster, Riegel and Rhoads[44] find the indirect evidence strong enough to state: "The feeding mixture should not contain too much fat, for even though the mixture is placed in the jejunum gastric emptying will be delayed. Failure to recognize this fact has greatly increased the time of convalescence of many patients."

The incidence of functional disorders of gastrointestinal motility after gastrectomy is hard to compute. In the first few months after operation, such disorders are doubtless quite prevalent, and they, as well as mechanical stomal defects, should be considered as possible causes of improper gastric evacuation after partial gastrectomy. The edema of hypoproteinemia, if present, may partially occlude the stoma, but it also produces a serious derangement of all gastrointestinal motor functions.[36, 38] With the aid of proper dietary management, functional disorders tend to subside during the first postoperative year, and subsequently they are encountered infrequently. Among 62 long-standing but poorly functioning gastroenterostomies — not gastrectomies — of various types, Eusterman, Kirklin and Morlock[45] found that only 6 could be ascribed to abnormal function.

The question naturally arises whether disorders of gastric emptying are particularly prevalent among those patients who experience postcibal distress after gastrectomy. Since the sudden inflation of a balloon in the duodenum often produces epigastric distress, nausea and giddiness,[46] some hold that these complaints occur after gastric resection because ingested food rapidly enters and distends the small bowel, particularly if the capacity of the gastric remnant is small or if total gastrectomy has been performed.[47, 48] This theory is compatible with the facts that the postcibal symptoms tend to subside during the first postoperative year and that during the same time new reservoirs are being created, either by enlargement of the gastric stump or by chronic dilatation of the upper small bowel. On the other hand, if the gastric remnant empties very slowly because of some mechanical or functional disorder, it is possible that symptoms of epigastric distress and fullness after eating are produced by abnormal intragastric tension. It must be admitted, however, that the rate of gastric evacuation after subtotal resection, as judged by roentgenologic observations, and the incidence of postcibal symptoms shows, at the most, a weakly positive correlation.[15, 16] Possibly functional disorders that are induced by the ingestion of certain foods are not evident after suspensions of barium sulfate are taken by mouth.

Absorption

Previous reviews[49, 50] on the effects of gastrectomy on absorption indicate that fat absorption is sometimes impaired, that fecal loss of nitrogen is excessive in a few scattered cases, but that the absorption of carbohydrates, so far as can be determined by available methods, is not decreased. Glucose administered intravenously is followed by a normal tolerance curve,[21] but the oral test yields a curve that shows certain deviations from the normal.[21, 32, 47, 51] In the gastrectomized patient, the blood-sugar level undergoes a steep rise, achieves a high peak and then drops rapidly to levels that may be definitely hypoglycemic. This curve probably depends on the facts that the resected stomach has little capacity to act as a reservoir and that the whole test dose enters the small intestine rapidly and is quickly absorbed. A strong stimulus is thereby exerted on the pancreas, which elaborates sufficient insulin to carry the blood-sugar to abnormally low levels after the absorption of glucose is completed. The secondary hypoglycemia may account for the weakness and perspiration experienced by some gastrectomized patients after taking a meal,[32] but opinions on this point differ.[47]

Intestinal digestion of protein apparently proceeds quite rapidly, even in the relative absence of gastric juice,[52] and oral glycine-tolerance tests indicate that amino acids are readily absorbed by patients with gastric resections.[53] In harmony with these observations is the fact that an abnormal loss of fecal nitrogen is most unusual in patients with subtotal gastrectomies. After total gastrectomy, Emery's[50] dogs showed some fecal loss of nitrogen, but only 1 out of 4 carefully studied human subjects with total gastrectomy suffered from an increased nitrogen output in the stools.[21, 23] Rekers, Pack and Rhoads[23] believe the nitrogen lost in this case to be of dietary origin, but there is considerable evidence that any process that increases the bulk of the stools may cause an increased fecal excretion of endogenous nitrogen.[54, 55]

Of the various processes of absorption, that of fats appears to be the one most easily affected by gastrointestinal disorders of all types. Earlier reports of steatorrhea after gastrectomy are found chiefly in the European literature, but recent work on this problem has been carried out in this country by Rhoads and his associates.[23, 56] These authors report that normal subjects on a diet of 80 gm. of fat a day lost 2 to 6 gm. of fatty material daily in the stools. In 3 patients with total gastrectomies, however, the fecal lipid loss was 11 to 23 gm. a day, although the daily fat intake was only 40 gm. The degree of steatorrhea was not relieved by giving bile salts, Lipocaic or intrinsic factor (Ventriculin), nor did it appear to depend on a lack of gastric chyme, a lack of reservoir function or an increased intestinal transport. In 2 of the 3 patients, however, the giving of excess protein by mouth appeared to

improve fat absorption, and in 1 patient steatorrhea was reduced by the oral administration of pancreatic enzymes.

The improvement attending the exhibition of pancreatic material caused Rekers, Pack and Rhoads[23] to suggest that gastrectomy may somehow interfere with pancreatic function. The pancreas has previously been suspected of being responsible for post-gastrectomy steatorrhea, but conclusive evidence on this point has been difficult to obtain. After gastric resection, the duodenal contents is said to contain normal amounts of trypsin and diastase[57] and urinary diastase is not significantly changed.[32] Atoxyl-resistant lipase in the serum, believed to be specific for lipase of pancreatic origin,[58] is reported to be increased in one series of gastrectomies[59] and not changed in another.[32] Santy and Mallet-Guy[16] state that a rather constant production of pancreatic lipase is stimulated in the normal subject after intraduodenal instillation of hydrochloric acid, but that the response in gastrectomized patients is extremely variable and unpredictable, even if the same subject is repeatedly tested. They therefore conclude that gastrectomy disorganizes the mechanisms that control the flow of the external pancreatic secretions. In evaluating contradictory reports on the effects of gastrectomy on pancreatic function, it is well to remember that the determination of pancreatic enzymes is difficult and often unreliable.

After total gastrectomy for cancer, prothrombin levels may not return to normal, but this abnormality cannot be ascribed to faulty absorption of the fat-soluble vitamin K, for even injection of the vitamin may not affect the low prothrombin levels.[24] When growing monkeys are gastrectomized, the serum calcium and phosphorus may be diminished and bony changes suggestive of rickets may develop, even though vitamin D is given.[17] In the past, Ivy[20] has ascribed disturbances in calcium metabolism in gastectomized animals to a lack of gastric secretions and to changes in the acid-base equilibrium, but his recent work with growing monkeys suggests that absorption of the fatty vitamin D in these animals may be deficient.

Anemia

No doubt exists that the normal stomach, in the digestion of beef muscle, forms some substance that relieves pernicious anemia,[60] but, after total gastrectomy, this disease does not appear in laboratory animals and occurs extremely rarely in man.[20, 61–63] Meulengracht[64] believed that he held the answer to this apparent contradiction when he demonstrated that in the pig the pylorus and the duodenum, not the fundus, contain the antianemic intrinsic factor. Subsequently, however, Meulengracht[65] was surprised to find that the stomach of patients dying of pernicious anemia showed extensive atrophy of the fundus and a normal appearance of the pyloric glands. The fallacy of Meulengracht's work lay, as Fox and Castle[66] showed, in applying his results in pigs to man; in the human stomach, the intrinsic factor is found principally in the fundus. The puzzle therefore still persists: Why do not patients with gastrectomies, particularly those with total gastrectomies, develop pernicious anemia?

As a matter of fact, a small number of patients do. In 1941, Meyer, Schwartz and Weissman[67] collected 54 cases of what they considered to be authentic cases of pernicious anemia following total gastrectomy. Since then another case has been reported,[68] but this case and others reported in the past have not had the benefit of a careful study such as Meyer and his associates devoted to their case. Before one can assert that true pernicious anemia occurs as a sequel to gastrectomy, control periods during which the patient receives extrinsic factor, reticulocyte counts before and during liver therapy, adequate follow-up periods and possibly even bone-marrow studies are necessary. Even if the evidence for pernicious anemia is unequivocal, the possibility still exists that some of the cases of pernicious anemia associated with gastrectomy are merely the result of coincidence.

Many patients and animals with gastrectomies do suffer from anemia, but this is usually of the normocytic and the normochromic[23, 24] or hypochromic variety.[21, 62, 69] The work of Dragstedt and his associates[70, 71] indicates that the gastrectomized dog remains anemic on a stock diet but responds well when given excess iron by mouth. Many post-gastrectomy anemias also respond to ferrous sulfate,[21, 62] but some remain refractory to both iron and liver.[23, 62] Although parenteral liver extract is recommended for treating post-gastrectomy anemias,[48] iron is apparently more valuable, either given alone or in conjunction with liver extracts. In addition, proteins and vitamin B complex and its specific fractions, such as riboflavin,[72] may also have to be given to excess if anemia after gastrectomy is to be prevented.

Petri and his associates[18, 73] believe that gastrectomy in young swine is followed by a symptom complex closely similar to pellagra. In man, pellagra secondary to gastrectomy has been reported,[74, 75] but in both cases the condition responded to adequate amounts of niacin (nicotinic acid) and vitamin B complex.

Summary

The consequences of removing portions of the human stomach depend on the nature of the primary disease, the extent of the resection, the surgeon's skill and divers factors that vary with the patient's age, diet and mental attitude. By and large, however, the following statements can be made concerning patients who have undergone subtotal or total gastrectomy:

About 30 per cent of the patients do not regain their normal weight, and some do not regain their preoperative weight.

About 10 per cent suffer from symptoms soon after taking a meal, particularly if the meal is large. These symptoms consist of epigastric distress and pressure, belching, nausea and weakness.

Some patients have soft stools, but very few suffer from persistent diarrhea.

The gastric remnant, which is at first small, gradually enlarges and resumes some of the reservoir function of the stomach. If the gastric remnant is extremely small, the small bowel dilates to form reservoirs near the anastomosis.

Even if the stoma is anatomically perfect, gastric evacuation after subtotal resection may be delayed by functional disorders.

The glucose-tolerance test shows a rapid rise in blood sugar, followed by a fall that may reach hypoglycemic levels. In a few cases symptoms occur during the hypoglycemic phase.

Carbohydrates and proteins are usually adequately absorbed, but some impairment in the absorption of fats may occur.

Pernicious anemia is an extremely rare sequela, but a normochromic or hypochromic anemia is not infrequent.

A nourishing diet, taken frequently in small quantities and fortified with vitamins and iron, often alleviates some of the complications of gastrectomy.

Symptoms and various motility disorders, which may be prominent during the first postoperative year, subsequently tend to correct themselves spontaneously.

Even when the undesirable sequelae of gastrectomy are marked, they rarely interfere with the patient's ability to work, and practically never are they so dangerous or distressing as the disease for which the gastric resection was undertaken. Hence the fact that these sequelae may occur cannot be considered in any sense as a contraindication to gastrectomy in adults. In the rare cases in which resection has to be considered in a growing boy or girl, it may be wise to limit the extent of the resection.

References

1. Walters, W., Gray, H. K., Priestley, J. T., and Counseller, V. S. Report of surgery of stomach and duodenum for 1941. *Proc. Staff Meet., Mayo Clin.* **17**:420-426, 1942.
2. *Idem.* Annual report on surgery of stomach and duodenum for 1942. *Proc. Staff Meet., Mayo Clin.* **18**:505-511, 1943.
3. Wangensteen, O. H., and Lannin, B. Criteria of acceptable operation for ulcer: importance of acid factor. *Arch. Surg.* **44**:489-500, 1942.
4. Wangensteen, O. H. Surgical problem of gastric cancer, with special reference to: (1) closed method of gastric resection, (2) coincidental hepatic resection, and (3) preoperative and postoperative management. *Arch. Surg.* **46**:879-906, 1943.
5. Miller, G. G. Report on 230 cases of subtotal gastric resection for peptic ulcer. *Surgery* **12**:383-387, 1942.
6. Marshall, S. F. Subtotal gastrectomy for ulcer: indications and technic. *S. Clin. North America* **22**:737-759, 1942.
7. Allen, A. W. Importance of antral mucosa in surgical treatment of duodenal ulcer. *South. M. J.* **36**:368-373, 1943.
8. Holman, C. W., and McSwain, B. Gastric acidity following gastric resection. *Surgery* **13**:916-923, 1943.
9. Friedell, M. T., Shaar, C. M., and Walters, W. Effect of gastric resection on gastric acidity. *J. A. M. A.* **120**:666-670, 1942.
10. Hollander, F., and Mage, S. Statistical method for evaluating results of treatment for peptic ulcer. *Surg. Gynec. & Obst.* **76**:533-546, 1943.
11. Church, R. E., and Hinton, J. W. Follow-up results in subtotal gastric resection for ulcer. *Am. J. Digest. Dis. & Nutrition* **9**: 317-321, 1942.
12. Browne, D. C., and McHardy, G. Patient after gastric surgery. *South. M. J.* **36**:32-41, 1943.
13. Gordon-Taylor, G., Hudson, R. V., Dodds, E. C., Warner, J. L., and Whitby, L. E. H. Remote results of gastrectomy. *Brit. J. Surg.* **16**:641-667, 1929.
14. Bremer, H., and Held, A. Beitrag zur Klinik und Röntgenologie des resezierten Magens. (Einfluss der Wirtschaftskrise auf die Erfolge der Magenchirurgie.) *Deutsche Zeschr. f. Chir.* **238**:466-484, 1933.
15. Strauss, A. A., Strauss, S., Levitzky, F., Scheman, L., Seidman, E. E., Arens, R. A., Meyer, J., and Necheles, H. Physiological and clinical study of patients after subtotal gastrectomy. *Am. J. Digest. Dis. & Nutrition* **4**:32-37, 1937.
16. Santy, P., and Mallet-Guy, P. Le déficit pondéral post-opératoire séquelle éventuelle de la gastrectomie pour ulcère. *Presse méd.* **47**:1-5, 1939.
17. Freeman, S., Hough, V. H., Wigodsky, H., and Ivy, A. C. Effect of gastrectomy upon growing monkeys. *Gastroenterology* **1**:199-210, 1943.
18. Petri, S., Norgaard, F., and Bing, J. Pathological changes produced by gastrectomy in young swine. *Am. J. M. Sc.* **195**:717-722, 1938.
19. Bussabarger, R. A., and Ivy, A. C. Hematologic studies on gastrectomized monkeys. *Proc. Soc. Exper. Biol. & Med.* **34**:151, 1936.
20. Ivy, A. C. Effects of gastrectomy in animals. *Am. J. Digest. Dis. & Nutrition* **7**:500-502, 1940.
21. Farris, J. M., Ranson, H. K., and Coller, F. A. Total gastrectomy: effects upon nutrition and hematopoiesis. *Surgery* **13**:823-833, 1943.
22. Joll, C. A., and Adler, D. I. Long survival after total gastrectomy: brief review, with report of two cases. *Brit. M. J.* **2**:632-635, 1942.
23. Rekers, P. E., Pack, G. T., and Rhoads, C. P. Metabolic studies in patients with cancer of gastrointestinal tract. VI. Disorders in alimentary digestion and absorption in patients who have undergone total gastrectomy for carcinoma of stomach. *Surgery* **14**: 197-215, 1943.
24. Pack, G. T., and McNeer, G. Total gastrectomy for cancer: collective review of literature and original report of twenty cases. *Internat. Abstr. Surg.* **77**:265-299, 1943.
25. Jordan, S. M. End results of radical surgery of gastrointestinal tract as seen by gastroenterologist. *J. A. M. A.* **126**:586-590, 1941.
26. Mateer, J. G. Discussion of Jordan.[25]
27. Vitkin, S. F. Motor functions of stomach after resection. *Ann. Surg.* **111**:27-48, 1940.
28. Weissenborn, W. Erfahrungen über 10jährige operative Behandlung von Magen- und Zwölffingerdarmgeschwüren unter besonderer Berücksichtigung der Fernergebnisse. *Arch. f. klin. Chir.* **195**:695-737, 1939.
29. Bloch, L. Discussion of Emery.[30]
30. Shay, H., and Gershon-Cohen, J. Experimental studies in gastric physiology in man: mechanism of gastric evacuation after partial gastrectomy as demonstrated roentgenologically. *Am. J. Digest. Dis. & Nutrition* **2**:608-613, 1936.
31. Brauch, F. Zur pathologischen Physiologie des resezierten Magens. *Klin. Wchnschr.* **18**:53-57, 1939.
32. Straaten, T., and Hünermann, M. Klinische Untersuchungen über die Blutzuckerregulation nach Magenoperationen. *Arch. f. klin. Chir.* **195**:62-105, 1939.
33. Schindler, R. Gastroscopic observations in resected stomachs. *Am. J. Digest. Dis. & Nutrition* **7**:505-507, 1940.
34. Hoag, C. L., and Saunders, J. B. Obstruction following gastroenterostomy or subtotal resection of stomach: treatment by jejunoplasty. *Arch. Surg.* **42**:259-278, 1941.
35. Mecray, P. M., Barden, R. P., and Ravdin, I. S. Nutritional edema: its effect on gastric emptying time before and after gastric operations. *Surgery* **1**:53-64, 1937.
36. Barden, R. P., Ravdin, I. S., and Frazier, W. D. Hypoproteinemia as factor in retardation of gastric emptying after operations of Billroth I types. *Am. J. Roentgenol.* **38**:196-202, 1937.
37. Ariel, I., Abels, J. C., Pack, G. T., and Rhoads, C. P. Metabolic studies of patients with cancer of gastrointestinal tract. XVI. Treatment of hypochloremia refractory to administration of sodium chloride, especially in patients with gastrointestinal cancer. *J. A. M. A.* **123**:28-30, 1943.
38. Leigh, O. C., Jr. Ileus associated with edema of bowel. *Surg. Gynec. & Obst.* **75**:279-284, 1942.
39. Chauncey, L. R., and Gray, H. K. Relationship of concentration of proteins in serum to postoperative gastric retention. *Gastroenterology* **1**:72-94, 1943.
40. Quigley, J. P. Modern explanation of gastric emptying mechanism. *Am. J. Digest. Dis. & Nutrition* **10**:418-421, 1943.
41. Abbott, W. O., Hartline, H. K., Hervey, J. P., Ingelfinger, F. J., Rawson, A. J., and Zetzel, L. Intubation studies of human small intestine. XXI. Method for measuring intra-luminal pressures and its application to digestive tract. *J. Clin. Investigation* **22**: 233-243, 1943.
42. Johnston, C. G., and Ravdin, I. S. Action of glucose on emptying of stomach: effect of varying concentrations in both normal stomachs and after various gastric operations. *Ann. Surg.* **101**:500-505, 1935.
43. Warren, R., Karr, W. G., Hoffman, O. D., and Abbott, W. O. Intubation studies of human small intestine. XV. Absorption and expulsion of glucose from stomach. *Am. J. M. Sc.* **200**:639-648, 1940.
44. Ravdin, I. S., Royster, H. P., Riegel, C., and Rhoads, J. E. Surgical care of patients with gastric cancer before and after operation. *Arch. Surg.* **46**:871-878, 1943.
45. Eusterman, G. B., Kirklin, B. R., and Morlock, C. G. Nonfunctioning gastro-enteric stoma: diagnostic study of sixty-two surgically demonstrated cases. *Am. J. Digest. Dis. & Nutrition* **9**:313-317, 1942.
46. Alvarez, W. C. *An Introduction to Gastro-Enterology.* 470 pp. New York: Paul B. Hoeber, Inc., 1940. P. 409.

47. Schwartz, A. H., Reingold, I., and Necheles, H. Investigation of relationship between blood sugar and general complaints following subtotal gastric resection. *Am. J. Digest. Dis. & Nutrition* **9**:151-154, 1942.
48. Lahey, F. H., and Marshall, S. F. Indications for, and experiences with, total gastrectomy. *Ann. Surg.* **119**:300-320, 1944.
49. Lublin, H. On late symptoms after gastroenterostomy and resection of stomach (Billroth II) for gastric and duodenal ulcer. *Acta Med. Scandinav.* Supp. **41**:1-131, 1932.
50. Emery, E. S., Jr. Cause of faulty digestion in dogs without stomachs. *Am. J. Digest. Dis. & Nutrition* **2**:599-608, 1936.
51. Lapp, F. W., and Dibold, H. Blutzuckerablauf in seiner Beziehung zum resezierten Magen. *Klin. Wchnschr.* **12**:547, 1933.
52. Elsom, K. A., Chornock, F. W., and Dickey, F. G. Intubation studies of human small intestine. XXIII. Method of determining digestive activity in any portion of gastro-intestinal tract, with some measurements of protein digestion in stomach and small intestine. *J. Clin. Investigation* **21**:795-800, 1942.
53. Ariel, I., Jones, F., Pack, G. T., and Rhoads, C. P. Metabolic studies in patients with cancer of the gastrointestinal tract. XII. Glycine tolerance test in patients with gastric cancer. *Ann. Surg.* **117**: 740-747, 1943.
54. Thaysen, T. E. H. *Non-Tropical Sprue: A study in idiopathic steatorrhoea.* 258 pp. London: Oxford University Press, 1932. P. 87.
55. Coffey, R. J., Mann, F. C., and Bollman, J. L. Influence of pancreas on utilization of foodstuffs. *Am. J. Digest. Dis. & Nutrition* **7**:144-149, 1940.
56. Rekers, P. E., Abels, J. C., and Rhoads, C. P. Metabolic studies in patients with gastrointestinal cancer. IV. Fat metabolism: method of study. *J. Clin. Investigation* **22**:243-248, 1943.
57. Stern, G. Untersuchungen über Magen- und Pankreasfunktion nach ausgedehnter Magenresektion. *Wien. klin. Wchnschr.* **42**: 1560-1563, 1929.
58. Lagerlof, H. O. *Pancreatic Function and Pancreatic Disease Studied by Means of Secretin.* 289 pp. New York: The Macmillan Company, 1942. P. 126.
59. Dibold, H., and Taubenhaus, M. Untersuchungen über das Verhalten atoxylresistenter Lipase nach Operationen am Magen. Ein Beitrag zur Frage der Pankreasfunktionsänderung bei Magenresezierten. *Klin. Wchnschr.* **12**:857-860, 1933.
60. Castle, W. B. Observations on etiologic relationship of achylia gastrica to pernicious anemia. I. Effect of administration to patients with pernicious anemia of contents of normal human stomach recovered after ingestion of beef muscle. *Am. J. M. Sc.* **178**:748-764, 1929.
61. Lublin, H. On anemia following gastric operations. *Am. J. Digest. Dis. & Nutrition* **3**:8-10, 1936.
62. Jones, C. M. Problem of gastrectomy and anemias. *Am. J. Digest. Dis. & Nutrition* **7**:502-505, 1940.
63. Sturgis, C. C., and Goldhamer, S. M. Macrocytic anemia, other than pernicious anemia, associated with lesions of gastrointestinal tract. *Ann. Int. Med.* **12**:1245-1262, 1939.
64. Meulengracht, E. Presence of antianaemic factor in preparations of dried stomach substance from cardia, fundus and pylorus respectively. *Acta Med. Scandinav.* **82**:352-374, 1934.
65. *Idem.* Histologic investigation into pyloric gland organ in pernicious anemia. *Am. J. M. Sc.* **197**:201-214, 1939.
66. Fox, H. J., and Castle, W. B. Observations on etiologic relationship of achylia gastrica to pernicious anemia. IX. Difference in site of secretion of intrinsic factor in hog and in human stomach. *Am. J. M. Sc.* **203**:18-28, 1942.
67. Meyer, K. A., Schwartz, S. O., and Weissman, L. H. Pernicious anemia following total gastrectomy. *Arch. Surg.* **42**:18-24, 1941.
68. Rhoden, A. J., and Grunbert, A. Macrocytic anaemia following partial gastrectomy. *Brit. M. J.* **1**:726, 1943.
69. Petri, S., and Jensenius, H. Experimental studies on production of pernicious anemia by operation on the digestive tract: survey of results of total gastrectomy and resections of stomach. *Acta Med. Scandinav.* **106**:274-292, 1941.
70. Mullenix, R. B., Dragstedt, C. A., and Bradley, J. D. Hemoglobin regeneration in gastrectomized dogs. *Am. J. Physiol.* **105**:443-449, 1933.
71. Dragstedt, C. A., Bradley, J. D., and Mead, F. B. Effect of iron on hemoglobin regeneration in gastrectomized dogs. *Proc. Soc. Exper. Biol. & Med.* **33**:58-60, 1935.
72. Spector, H., Maass, A. R., Michaud, L., Elvehjem, C. A., and Hart, E. B. Role of riboflavin in blood regeneration. *J. Biol. Chem.* **150**:75-87, 1943.
73. Petri, S., Norgaard, F., and Bandier, E. Studies on effect of nicotinic acid upon experimental gastroprival pellagra. *Acta med. Scandinav.* **98**:117-127, 1938.
74. Simpson, S. L. Secondary pellagra. *Quart. J. Med.* **4**:191-201, 1935.
75. Bean, W. B., Spies, T. O., and Blankenhorn, M. A. Secondary pellagra. *Medicine* **23**:1-77, 1944.

NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-THIRD ANNIVERSARY

House of Delegates, May 15 and 16, 1944

THE House of Delegates convened at the Hotel Carpenter, Manchester, on May 15, 1944, at 7:30 p.m., with Speaker George F. Dwinell presiding.

The following members answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
P. R. Hoyt, Laconia (alternate for Benjamin W. Baker)
Richard W. Robinson, Laconia
W. J. Paul Dye, Wolfeboro
Francis J. C. Dube, Center Ossipee
A. C. Johnston, Keene
Richard C. Batt, Berlin
R. N. Jones, Whitefield (alternate for Leander P. Beaudoin, Berlin)
Arthur B. Sharples, Groveton
Arthur K. Burnham, Lebanon
Leslie K. Sycamore, Hanover
Howard N. Kingsford, Hanover
Donald M. Clark, Peterborough
Stillman G. Davis, Nashua
R. E. Biron, Manchester
Deering G. Smith, Nashua
Clinton R. Mullins, Concord
William P. Clough, Jr., New London
Gerard Gaudreault, Concord
Willard C. Montgomery, Epping
Harry B. Carpenter, Portsmouth
Fred Fernald, Nottingham
Roland J. Bennett, Dover
George G. McGregor, Durham
B. Read Lewin, Claremont
Donald G. Moriarty, Newport

The Speaker declared a quorum present, and appointed the Credentials Committee as follows: Drs. Dube (chairman), Carpenter and Lewin. Dr. Dube reported that the credentials were in order.

The Speaker appointed the Committee on Officers' Reports as follows: Drs. Mullins (chairman), Smith and Montgomery. He appointed the Committee on Communications and Memorials as follows: Drs. Kingsford (chairman), Sharples and Bennett. To the Committee on Nominations, he appointed Drs. Dye (chairman), Robinson, Burnham, Clark and Bennett.

On motion duly made and seconded, it was voted to omit the reading of the previous minutes, because of the publication of the proceedings.

On motion duly made and seconded, it was voted to dispense with the reading of the reports of the councilors, since they will be published in the proceedings.

The secretary-treasurer, Dr. Carleton R. Metcalf, presented his report, as follows:

Membership, December 31, 1943

PAID

Belknap County	23
Carroll County	13
Cheshire County	20
Coos County	25
Grafton County	53
Hillsborough County	104
Merrimack County	57
Rockingham County	51
Strafford County	29
Sullivan County	12
Not in county society	3
	390

UNPAID

Affiliate members	25	
Honorary members	6	
Members in service	114	
	145	
		535

The total membership on December 31, 1942, was 551.

Financial Statement

RECEIPTS

January 1, 1943—balance forward	$248.12
Belknap County	168.00
Carroll County	90.00
Cheshire County	126.00
Coos County	168.00
Grafton County	318.00
Hillsborough County	612.00
Merrimack County	366.00
Rockingham County	306.00
Strafford County	168.00
Sullivan County	78.00
Net receipts, 1943 annual meeting	14.99
Cash received at annual meeting	56.00
Members not in county societies	24.00
Refund (Cancer Commission)	9.20
Interest on bonds (received from trustees)	37.50
Women's Auxiliary	25.00
Donations to National Physicians' Committee	191.00
Full subscriptions to *New England Journal of Medicine*	12.00
General Fund (for Procurement and Assignment)	359.66
General Fund (for Wagner Bill, Blue Shield and general expenses)	1000.00
	$4377.47

EXPENDITURES

New England Journal of Medicine:	
Journals	$495.11
Full subscriptions	12.00
Transactions	479.41
Carleton R. Metcalf (salary)	400.00
Printing	61.00
Envelopes and stamps	39.67
Eagle and Phoenix Hotel Company (committee lunches)	78.40
Halftone cuts	30.85
Clerical work	163.00
Telephone and telegraph calls	42.03
Mimeographing	26.25
Gold medal	16.75
Frank J. Sulloway (retaining fee, 1941 and 1943)	200.00
Dr. Hsieh (fee as guest speaker)	75.00
Lee and Cope (expenses of guest speakers)	9.09
George C. Wilkins (Cancer Committee)	50.00
Harris E. Powers (expenses at Congress of Industrial Health)	129.02
Deering G. Smith:	
Medical Preparedness expenses	359.66
Dues collected at annual meeting	14.00
Expenses at American Medical Association meeting	141.98
Merrimack Co. and Center district societies (dues collected at annual meeting)	28.00
Leander P. Beaudoin (dues collected at annual meeting)	7.00
Frederic S. Gray (dues collected at annual meeting)	7.00
Benevolence Fund:	
Interest on bonds	37.50
Women's Auxiliary	25.00
$1.00 per each paid member	404.00
National Physicians' Committee (donations):	
Coos County	36.00
Carroll County	13.00
Grafton County	75.00
Merrimack County	1.00
Rockingham County	38.00
Strafford County	28.00
Madeline A. May (stenographer at annual meeting)	167.62
Blue Shield and Wagner Bill (expenses of meetings and luncheons)	412.07
Mechanics Bank (service charges)	1.64
Flowers (funeral of Mrs. Dennis E. Sullivan)	5.00
	$4109.05
Balance, January 1, 1944	268.42
	$4377.47

The Benevolence Fund on December 31, 1943, amounted to $2572.78. Of this amount the principal was $2081.83 and the accrued income $490.83, part of which was from interest on United States bonds. During the past year we have received $25 for this fund from the Women's Auxiliary of Merrimack County.

Our receipts have again been curtailed because members who are in the Service do not pay dues and because we have increased the allotment for the Benevolence Fund to $1 from each member. We have had to draw on the General Fund to make up this deficit. We could decrease our expenses a little for the coming year, but there are only two items in our budget that would make an appreciable difference if they were eliminated. The first item is the *New England Journal of Medicine*. The *Journal* is sent to each member once a month and costs the Society between $400 and $500 annually. The second item is the Benevolence Fund. According to a vote of the House of Delegates this fund is to accumulate until it reaches $10,000; the interest from the fund may then be used to help needy members of the Society. It will be several years, therefore, before this fund becomes effective, and even then the available interest each year will be relatively small. You may wish to discontinue the annual allotment toward this fund for the duration of the war.

We have already economized in our expenses by eliminating a donation of $100, which for several years was given annually to the Women's Auxiliary, by limiting the annual meeting to one day and by choosing most of our guest speakers from nearby states rather than from distant ones.

Dr. Clarence O. Coburn, of Manchester, died on March 30, 1944. He was a former president of the Society and at the time of his death was a trustee, a councilor from Hillsboro County and a member of the Committee on Tuberculosis.

A year ago the House of Delegates made the following recommendations, which have been carried out:

Fifty dollars was given to the Cancer Committee for its work.

Your president and secretary have conferred with the State Welfare Department, with the result that the fees for old-age assistance cases have been increased. In accordance with the requirement of the federal government, however, the bills of the doctor must still be sent to the recipient and not to the Department of Welfare.

I asked each county society to have its members contribute $1 apiece to the National Physicians' Committee. During 1943, $191.00 was received from the several counties, as follows:

Carroll County	$13.00
Coos County	36.00
Grafton County	75.00
Merrimack County	1.00
Rockingham County	38.00
Strafford County	28.00

In accordance with your directions the Committee on Medical Economics, with the assistance of the three officers of the Society and of Mr. Sulloway, formulated a plan for a medical-service corporation. Two special meetings of the House of Delegates were held to consider this plan as it developed, one in September and another in November. The Committee on Medical Economics will give you the intimate details in the report that they are making at this meeting.

Several members of the Society have discussed the Wagner Bill before county medical societies, Rotary Clubs and other organizations and have emphasized the fact that the Society has expressed its opposition to this plan for socialized medicine. The Wagner Bill will probably come to the fore in Washington within the next few months. It is still a very definite threat to the present system of the practice of medicine.

We must watch carefully House Bill 352, which was introduced into the New Hampshire Legislature at its recent special session and which was referred, without discussion, to the General Court, which assembles next January. This is a bill "relating to Sickness and Non-Industrial Accident Compensation." It is apparently a slightly modified Wagner Bill that is to be administered not by the federal government but by the State of New Hampshire.

In the several county societies during the past year there has been animated discussion of the Emergency Maternity and Infant Care Program for the families of servicemen, which was promulgated by the Children's Bureau in Washington. This matter was taken up at our meeting a year ago but no definite recommendation was made. A motion was carried that the "House of Delegates consider the Government plan for the payment of hospital and medical care of servicemen's wives at the time of delivery." It was pointed out that many of these patients were able to pay more than the $35 fee that is provided by the Government, and a suggestion was made that the Government pay this money directly to the patient and that the doctor arrange a suitable fee in each case as he would with other private patients. It is doubtful, however, whether the Children's Bureau will accede to any such suggestion. In California the state medical society has rejected the plan of the Children's Bureau and has voted to treat these women as service cases.

Here in New Hampshire the present status of this program has been summarized as follows:

By and large our physicians are not enthusiastic about the program.

The following are the reasons for the reluctance; the first is the reason most frequently given:

The great fear that this program is an entering wedge for some type of government-controlled medicine.

The seemingly dictatorial way in which this program has been set up; the physician must accept the patient at the fee established by the program and cannot further charge the patient. There is no financial investigation, regardless of the financial status of the patient.

The over-all fee of $35, which is regardless of the complications that may arise. This fee was set by the Maternity and Infancy Committee of the New Hampshire Medical Society. No state has a fee of more than $50, and that is the maximum allowed by the Children's Bureau. Maine, New Hampshire and Vermont, comparable states, have a fee of $35. Massachusetts has a fee of $50. Physicians cannot understand why cases should not be treated individually. There is the fear that if such a program were here to stay, the physicians would be limited to $35 and people would begin to think that that was all an obstetric case was worth.

However, most physicians doing obstetrics are participating in this program even though some of them may not be in accord with it, for several reasons:

Pressure from patients or public opinion.

This is an "all-or-none" program and takes care of both hospital and medical bills; if the physician does not sign the application blank, the patient cannot get hospitalization, so the physician, rather than penalize the patient, signs the blank and accepts the patient. This kind of regulation puts pressure on the physician to sign the application. Physicians object to this kind of pressure.

It should be noted that during the coming year the Children's Bureau is asking for seven prenatal examinations of the mother under the present fee schedule; that it is authorizing additional payment for "major non-obstetric surgical operations needed during pregnancy and six weeks postpartum for illness not attributable to pregnancy" at a rate not to exceed a total of $50 for preoperative, operative and postoperative care; and that it is authorizing additional payment during pregnancy and six weeks postpartum for the home or hospital treatment of "intercurrent conditions not attributable to pregnancy which do not require major surgery." For this care the fee is not to exceed $12 for the first week of illness and $6 a week for subsequent weeks of illness. Similar authorizations with the same fees are made for an infant during its first year.

I have asked the Committee on Maternity and Infancy to consider this plan again at the present session and to advise whether or not members of this Society should continue the service under the present arrangement.

Because our program is crowded we have omitted the usual president's address and the report of the trustees. The report of the trustees will, however, be printed in the *New England Journal of Medicine* in a month or two and will be incorporated in the *Transactions* next year.

CARLETON R. METCALF, *Secretary-Treasurer*

On motion duly made and seconded, it was voted that the report of the Secretary-Treasurer be referred to the Committee on Officers' Reports.

Dr. Mullins, chairman of the Committee on Officers' Reports, then spoke as follows:

The Committee on Officers' Reports wishes to congratulate the Secretary-Treasurer on his successful attempt to curtail expenses. We recommend that the contribution to the Benevolence Fund be suspended for this year. The possibility of making this up later by a transfer from the General Fund is suggested. It is thought that the funds for the *New England Journal of Medicine* should be continued, since it serves a rather important purpose. The other suggestions as to Maternity and Infancy are dealt with in the respective committee reports.

Dr. Peters moved the adoption of this portion of the report, and this motion, duly seconded, was carried.

Report of the Committee on Maternity and Infancy

This is the tenth year of the study of maternal and infant deaths.

MATERNAL DEATHS

There were 24 maternal deaths in 1943. There were 9797 live births, thereby giving a maternal mortality of 2.45 per 1000 live births. This may be compared with the figure for 1942, when there were 15 maternal deaths and 9599 live births, a maternal mortality of 1.6 per 1000 live births.

These 24 maternal deaths have been classified according to the *International List of Causes of Deaths* as follows:

Classification	No. of Deaths
147 (*d*) Puerperal embolism (during childbirth and puerperium)	5
146 (*c*) Hemorrhage of childbirth and puerperium as placenta accreta, post partum	4
146 (*a*) Hemorrhage — placenta previa with childbirth	2
148 (*a*) Puerperal toxemia (death after delivery)	2
149 (*b*) Complications following cesarean section	2
146 (*b*) Hemorrhage — premature separation of placenta with childbirth	2
147 (*b*) Infection during childbirth and puerperium	2
143 (*b*) Hemorrhage — premature separation before delivery	1
144 (*d*) Toxemia of pregnancy (death before delivery)	1
149 (*a*) Accidents of pregnancy — inversion of uterus	1
140 (*b*) Abortion with infection	1
148 (*b*) Puerperal toxemia (albuminuria and nephritis)	1
Total	24

The committee has reclassified these for the purpose of this report in the following way:

Cause of Death	No. of Deaths	
Accidents of pregnancy		6
Puerperal embolism	5	
Inversion of uterus	1	
Hemorrhage		9
Placenta previa	2	
Premature separation	3	
Retained placenta	1	
Uterine inertia	3	
Toxemia (all types)		4
Puerperal sepsis		4
Abortion with infection		1
Total		24

The 14 cases reviewed were listed on the death certificates as follows:

Embolism; endometritis (G6)
Hemorrhage and shock; premature separation of the placenta; death before delivery (G2)
Postoperative shock and hemorrhage; placenta accreta (G3)
Eclampsia; pregnancy with toxemia (G10)
Post-partum hemorrhage and shock; placenta previa complete (G4)
Embolism of lungs (acute schizophrenia) (G24)
Embolism; endometritis (G6)
Toxemia of pregnancy; acute nephritis (G11)
Hemorrhage; placenta previa (G13)
Coronary thrombosis following childbirth (G14)
Premature separation of placenta with shock and hemorrhage (G17)
Post-partum hemorrhage (G18)
Peritonitis and intestinal paralysis following cesarean section (G19)
Eclampsia (G21)

The 10 cases not reviewed were listed on the death certificates as follows:

Inertia of uterus with uterine hemorrhage
Toxemia of pregnancy
Purulent endocarditis; several thrombi; atelectasis of lung; cesarean section
Pulmonary embolism; difficult delivery
Inversion of uterus
Pulmonary embolism; cesarean section
Shock; abruptio placentas; hydrocephalic breech
Cardiac collapse and generalized toxemia; penetration of uterus by piercing instrument; post-abortion uterus
Acute dilatation of stomach due to intestinal obstruction following operative delivery; acute peritonitis
Post-partum hemorrhage

Of the cases reviewed, in Cases G2, G3, G4, G6, G13 and G18 more information was needed in order to classify them. Cases G10, G17 and G21 were classified as due to the fault of the patient; Cases G11 and G24 were unavoidable; and Cases G14 and G19 were probably avoidable.

Infant Mortality

Included in this group are those infants who died under one year of age. There were 420 infant deaths in 1943, giving an infant death rate of 42.9 per 1000 live births, compared with a rate of 33.5 in 1942. The causes of death were as follows:

Cause of Death	No. of Deaths
Deaths occurring during the first day:	
Prematurity	90
Birth injuries	22
Congenital deformities	15
Atelectasis	7
Congenital debility	2
Cerebral hemorrhage	1
Bronchopneumonia	1
Pulmonary emphysema	1
Syphilis	1
Unknown	1
Total	141
Deaths occurring during the first month exclusive of the first day:	
Prematurity	44
Congenital deformities	36
Birth injuries (all kinds)	9
Atelectasis	7
Diseases of gastrointestinal tract (diarrhea, enteritis and so forth)	7
Pneumonia (all types)	11
Hemorrhagic disease of newborn	1
Diseases of thymus gland	3
Congenital debility	1
Acute nephritis	1
Unknown	8
Total	128
Deaths occurring from the second to the twelfth month, inclusive:	
Pneumonia (all types, with complications — empyema and so forth)	56
Congenital malformations	24
Gastroenteritis	19
Influenza	12
Whooping cough	6
Pyelonephritis	3
Congenital debility	8
Birth injury	2
Suffocation	5
Atelectasis	2
Tuberculosis (pulmonary)	1
Meningitis	1
Cancer of intestines	1
Endocarditis	2
Otitis media	1
Mental deficiency	1
Convulsions	1
Unknown	5
Total	151

The causes of 211 still births were as follows:

Cause of Still Birth	No. of Cases
Stillborn (cause unknown)	42
Intrauterine death (cause unknown)	15
Toxemia	14
Difficult delivery	17
Asphyxia neonatorum	28
Prematurity	24
Congenital malformations	42
Premature separation of placenta	22
Placenta previa	3
Erythroblastosis	1
Ruptured uterus	2
Total	211

The committee, with the aid of Dr. Burpee, the consultant, has formed the following recommendations in regard to the Emergency Maternity and Infancy Care Program.

That the physicians of New Hampshire, as a part of their war effort, participate in the maternity program in the future as they have done during the past year.

That the fee for services under this program be increased to $50 per case.

That the program suggested for additional care for major surgical and medical conditions occurring in the course of pregnancy *not* be accepted. (This recommendation is made because of the difficulty involved in determining the meaning of the term "qualified consultants" as demanded under the program.)

That the child-health program be accepted under the following conditions:

The major surgical aid not to be included (again because of the difficulty in determining the meaning of the term "qualified consultants").

The medical aid to be accepted for illnesses requiring *over* two home or office visits. (This plan would eliminate many needless calls on essentially well babies, and would avoid a great deal of extra work involved in applying for aid, authorizing the service and approving the records for payment.

Marion Fairfield, *Chairman*
John J. Boardman
Benjamin P. Burpee, *Consultant*

Dr. Clough then spoke as follows:

I think there is not one of us who is not perfectly willing to do anything we can for servicemen's wives, but we are unwilling to have this thing forced down our throats.

Why should a physician be forced to sign the application blank, knowing that if he refuses to do so the patient will receive absolutely nothing? That is one outstanding objection I have to the program. If the physician does not sign this slip of paper and give the patient the $35 delivery, she gets nothing.

I see absolutely no reason for this clause's being inserted. The number of pitfalls that a man can find himself in under this program is great. I have one case that could have been much worse. A young girl was in her first month of pregnancy, and in the second month, she started to miscarry. She lived nine miles from the office. I saw her in the middle of the night for two hours, and brought her to the hospital by ambulance. I kept her in the hospital for ten days and took her home myself. I made nine house calls. She happened to be a girl who bled every time her period was due. I saw her in the office eighteen times. In the seventh month of pregnancy, she had a premature labor and delivered a 2½-pound infant who, for some reason or other, made the grade; the mother developed cystitis. We discharged her on the fifteenth day. I had to make three calls to see her after she got home. We kept the baby in the hospital for eight weeks. This particular case could even have been worse, but it is a good example of what we are letting ourselves in for if we sign this blank.

There is also a tremendous difference between putting the program into effect in the cities and in the country towns. Those who practice in the cities can put these patients on ward service, but in the country we have to take care of them and call on them in their homes, no matter where they live.

The whole program has failed in its purpose, too. It would be interesting to note how many obstetricians in the cities are actually taking cases, and how many women are actually getting the care they want. I doubt if many of the obstetricians are taking them. They are going around, hunting for some doctor to take them for $35, with the result that they get somebody who is not too good an obstetrician.

Dr. Gaudreault said that he was in perfect agreement with Dr. Clough. He thought that the forms should be submitted to the superintendents of hospitals and should be signed by them and that the doctors should be allowed to do what they please. He said that he would much rather deliver a case for nothing than be paid $35 for his services, and believed that if anyone was willing to sign a paper he should receive at least $50, as in Massachusetts and several other states. He recommended two different forms — one for the hospital and one for the physician.

Dr. Dube said that the superintendent of his local hospital was seriously disturbed by the whole affair. Apparently some time ago the hospital was offered a lump sum of $50, but the option was then withdrawn and the hospital was asked to find the per capita cost for keeping a maternity patient. A detailed statistical report was wanted covering the number of patients in the hospital, the number of private rooms, the number of nurses on hand, the exact cost of food per meal and everything else. Apparently the hospital was being offered not over 85 per cent of the per capita cost of the care of the patient. Dr. Dube asked for information from the committee on the matter.

Dr. Smith then read the following extract from his report as delegate to the meeting of the American Medical Association:

The action of the federal government in making funds available for maternity and infant care for the wives and infants of enlisted men was approved and adoption was urged of a plan under which the federal government will provide for the wives of enlisted men a stated allotment for medical, hospital, maternity and infant care similar to the allotments already provided for the maintenance of dependents, leaving the actual arrangements with respect to fees to be fixed by mutual agreement with the wife and the physician of her choice. This action was similar to that taken by the New Hampshire House of Delegates last May. A copy of the resolution was sent to each congressman and each senator urging that a method of making available these funds be changed to an allotment basis. After considerable discussion in the House of Representatives, the proposal was rejected by a vote of 115 to 8.

He spoke as follows:

My feeling is that medical care is no different than the provision of fuel, food and shelter and clothing for the wives and children of the servicemen, and that medical care should be included in the allotments or allowances now given to servicemen. If the allowances, as they are called, are not large enough to take care of the wife and the children when they are sick or when the wife is going to have a baby, they should be increased. They are, as you know, a matter of record, varying according to the number of children a soldier has. If there are no children, the wife receives $50 a month from the Government; if there is one child, she receives $80 a month; if there are two children, she receives $100, and there is an increase of $20 for each additional child. Of this money, $22 comes from the soldier's pay and the balance is made up by the Government.

Much as we want to take care of the soldier's wives, why should we do it at a cut rate when the Government is paying the full rate for other essential services?

Dr. Mary Atchison spoke as follows:

I am here neither to defend nor to sell. I feel, however, that the time has come for this controversial subject to be discussed frankly and openly. We in the State Health Department who have been delegated with the job of trying to administer this formidable program find ourselves in the middle, so to speak.

The fact is that funds are made available through the program by an act of Congress for the care of the specific group of individuals, namely, the wives and prospective mothers of servicemen, and up to this date there is no other source of funds for the women.

Medical care, to be sure, is absolutely no longer a luxury; it is a necessity, and it should be so classed as much as is food, clothing or shelter, as Dr. Smith has stated.

If the wife of a serviceman becomes ill with any other condition than pregnancy, no provision is made for her. Therefore, this program is, as designated, an emergency maternity program, and infant care is included in other states. We have been promised that the program is for the duration of the war and for six months thereafter, only. I think we should safeguard our rights in this regard.

I happened to be present at the state health officers' meeting in Washington when this matter was frankly discussed. The state health officers told the Children's Bureau officials exactly what they thought in regard to the manner of administration, the red tape, the requirements and the restrictions imposed in the administration of this program.

By and large, the physicians in New Hampshire have co-operated beautifully. They have given their time. They have, without liking it, given their services, and these have been of the best. We are grateful to you for doing that.

We sent out a bulletin to you before this meeting, and enclosed with it a short explanatory note. The bulletin had a formidable outline. We asked you to read and study it, so that you would know what the proposals or the policies are. We asked you in particular to note Item 4, which describes the services that are available under

the program. Your Committee on Maternity and Infancy has studied this program with a great deal of care and albeit, after much frank discussion, they do not approve of all of it. It is within our rights to prepare a program, provided you will accept it in principle advising us as to the parts of the program that you think might be workable. We are asking for a ceiling fee for maternity care of $50, and if this is accepted by you I am almost certain we shall get it. The reason we have not asked for $50 before is that when the program was first started, the ceiling set by the Bureau was $35. Gradually, the various states studying the matter reached the conclusion that a maternity case could not be handled for $35. It was stated that if that was all such a case was worth, the physicians were not going on record as saying that they would lower their standards in order to give a $35 delivery. So Massachusetts and some of the larger states asked for $50 and got it. I understand from my colleagues in Vermont and Maine that they are going to do the same.

In the beginning, in such a case as Dr. Clough described, we were not allowed to make any additional provisions. As stated in the bulletin we shall be allowed to pay an additional amount for time and travel on a mileage basis. We do not like these limitations any more than you do. These policies were worked out by the Children's Bureau with its medical advisory committee, which is made up of some of the best minds in the country — representatives of the American Medical Association, obstetricians, pediatricians and men recognized in the field of medicine as the best.

This is a mass-production type of program. It was designed, of course, to fit the United States as a whole. We cannot possibly say that one plan is going to fit the whole situation. From letters that we have received we have assurances that we can use our own judgment.

I want to compliment Dr. Chamberlain for the conscientious manner in which she has tried to administer the program. I am not saying whether or not it should be adopted, but I want you to know that we are constantly aware of the needs of the profession and its problems, and our only purpose is to try to protect its interests so far as a program of this type is concerned.

When this act was passed by Congress there must have been some reason for it, and this was pressure from certain groups. The American Red Cross, all the labor unions and others simply went to Washington and demanded that something be done.

As I understand it, the entire intent of the program is a service to the serviceman and his family.

The matter of the payment of funds to the family was also discussed. It was thought that the most democratic thing to do was to give the patient a sum of money and say to him or her, "Pay your bill." However, we in the Department of Public Welfare, who administer the public-relief moneys, know that the grocer gets paid, the furniture dealer gets paid, the dry-goods store gets paid, but the doctor does not get paid. When the patient gets the money, there is nothing that says, "You must pay the doctor." Human beings are human beings; therefore, from an administrative standpoint, it is much better, in providing services, in order to safeguard everything, that the doctor be paid by an official agency. The official agency is the department that has in it medical personnel. The only reason that the payment is made in this way is that the doctor can thus be sure of what he is getting.

Dr. Mullins asked whether if this program were rejected it would work a real hardship on the women concerned in this state.

Dr. Atchison replied that, although she had no statistics to bear out her statement, the program was needed. The Grenier Field Army Base, for instance, had previously taken care of the Army wives, but had now discontinued this service which meant that the women with the $50 allotment were put on their own and would have to obtain care. She did not see why the doctor should "take the rap," since the patients had to pay the hospitals.

Dr. Mullins asked whether the hospitalization part of the infant-care program was connected with the physician's care, and whether the hospital care would still be allowed.

Dr. Atchison believed that the principle was the same, and Dr. Chamberlain said that it was the same setup. Dr. Atchison explained that the intention was that the patient should be assured of medical care of some sort, and of course would get hospital care, too. The hospital would be paid on a per diem ward cost basis, which would vary with different hospitals. This cost was to include diagnostic procedures, x-ray examinations, laboratory fees and what not.

Dr. Dube pointed out that this cost was nearly $6 a day. Dr. Atchison agreed, and remarked that if a maternity case was complicated, the physician would take a loss, but that if he had 100 cases and the majority of them were normal, he would not fare too badly. She added that, as a matter of fact, many physicians in the rural areas seemed to favor this program more than one would suspect. The city physicians, of course, had a higher overhead and they claimed that $35 was nothing but that $50 was a little better. However, in some states the physician got at the most $15 for a delivery, and even then could not collect it, so that many of them were willing to handle the case on the present basis.

Dr. Clough said that it depended on what type of obstetrics one was practicing. He believed that the price for all should be elevated, not brought down. It seemed to be the accepted attitude that obstetrics was not worth much, but this idea was absolutely wrong.

Dr. Atchison agreed. She added that of course it could not be said that one physician was worth $100 and another $25 and still others $15 or $50, so that a uniform method was the only possible one for proper administration of payments.

Dr. Dye asked whether women who accept ward-rate care could have private rooms. Dr. Atchison replied, "No."

Dr. Gaudreault said that he still believed that the doctor and the superintendent of the hospital should both sign the form, and have the doctor free to give the patient the care she needed and still not deny her the privilege of having her hospital care paid for by the Government.

Dr. Atchison spoke as follows:

> Under certain conditions under which these services are made available, Dr. Chamberlain and I see a loophole. For instance, these services may be authorized, provided similar services are not available. Suppose there is not a service in the hospital and that there is a clinic available in a certain town to which the patient can go. If she so chooses, all right. A wife has three choices under the program, including all types of available facilities. She may not want to go to a clinic, and may prefer a private practitioner and ask for one.

By the way there is nothing compulsory about this. The program is accepted by the medical society in principle. If you do not want to participate in it, you can say that you are too busy, that you would like to do it but cannot. That is your problem.

Dr. Gaudreault remarked that the program still left the doctor "holding the bag."

Dr. Atchison replied that under the maximum of $35 the doctor "was holding the bag," and that $50 was a little fairer. She thought that if everyone asked himself, "How much do you actually collect?" he would admit that it would be somewhere in the vicinity of $50.

The Committee on Officers' Reports moved that this program as recommended by the Committee on Maternity and Infancy be accepted. He outlined these recommendations as follows:

That the physicians of New Hampshire, as a part of their war effort, participate in the maternity program in the future as they have done during the past year.

That the fee for services under this program be increased to $50 per case.

That the program suggested for additional care for major surgical and medical conditions occurring in the course of pregnancy be not accepted.

This motion was duly seconded and was carried with three dissenting votes.

(To be continued)

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

BENJAMIN CASTLEMAN, M.D., *Acting Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 30351

PRESENTATION OF CASE

First admission. A fifty-nine-year-old housewife entered the hospital because of "heart trouble."

The patient had been in apparent good health until about three or four months before entry, when she developed chills, fever, chest pain and cough, diagnosed and treated as pneumonia by her physician. She recovered in about one month, but remained in bed except for about two or three hours daily. About two months before admission she had a sudden attack of agonizing, squeezing pain in the chest associated with inability "to catch her breath." She said that she felt as though she were "dying." The pain and dyspnea lasted for five hours and were finally relieved by a hypodermic injection by her physician. From that time on, she had attacks of nocturnal dyspnea occurring two or three times a week, which were relieved by injections, the nature of which is not recorded. She had marked orthopnea, requiring three pillows. One month before entry she developed frequent attacks of nausea and vomiting.

At forty-one years of age, following an episode of polydipsia, polyuria and weakness, she was found to have diabetes. The diet was regulated and she was placed on 4 units of insulin three times a day. Four months before admission the insulin was reduced to 4 units twice a day. Since no sugar appeared in the urine, the insulin was not increased during the course of the pneumonia, and one and a half months before entry it was stopped completely without any symptoms. One year before admission she experienced swelling of the ankles, and about six months later was found to have a systolic blood pressure of about 200. The past history was otherwise negative.

Physical examination showed a well-developed but poorly nourished dyspneic woman. The heart was enlarged. The apical impulse was palpable in the anterior axillary line. The sounds were strong, labored and regular. The first sound was clear. No definite murmurs were heard. The aortic second sound was greater than the pulmonic. The liver edge was palpated two fingerbreadths below the costal margin. There was moderate sacral and ankle edema. Ophthalmoscopic examination showed irregular thickening of the arterioles and the silver-wire reflex, as well as a number of small flame-shaped hemorrhages at the branching of the vessels. Foci of old hemorrhage and spots of exudation were present.

The blood pressure was 254 systolic, 100 diastolic. The temperature was 100°F., the pulse 90, and the respirations 20.

Examination of the blood showed a red-cell count of 2,000,000, with 6 gm. of hemoglobin. The white-cell count was 6600, with 60 per cent neutrophils. A blood Hinton test was negative. The urine was slightly cloudy, with a specific gravity of 1.012 and a pH of 7.5; it gave a +++++ test for albumin but no reaction for sugar, diacetic acid, acetone or bile. One or 2 red cells and 3 or 4 white cells per high-power field were seen in the sediment. A culture yielded *Staphylococcus albus* and colon bacilli. Stool examination was negative.

An x-ray film of the chest showed an enlarged heart whose borders, however, were obscured by fluid in the pleural cavities. The pulmonary markings were prominent. The nonprotein nitrogen was 77 mg. per 100 cc., the protein 4.8 gm., and the

*On leave of absence.

blood sugar 123 mg. The carbon dioxide was 20.6 millimols per liter, and the chloride 103.2 milliequiv. The fluid intake was limited to 1800 cc. She was given 0.011 gm. (⅙ gr.) of morphine and 0.2 gm. (3 gr.) of digitalis three times a day for two days and 0.1 gm. (1½ gr.) of the latter daily thereafter, as well as 1 gm. (15 gr.) of ammonium chloride daily. After the second hospital day she was started on protamine zinc insulin, 20 units daily for two or three days.

During the first hospital week she vomited considerably and complained of subxiphoid pain. The vomiting finally stopped. On the seventh hospital day the nonprotein nitrogen was 90 mg. per 100 cc. The protein was 6.0 gm. per 100 cc., with an albumin-globulin ratio of 2. The carbon dioxide was 13.2 millimols per liter, and the blood chloride 113.8 milliequiv. She was given 0.13 gm. (2 gr.) of sodium phenobarbital subcutaneously three times a day. A phenolsulfonephthalein test gave less than 5 per cent excretion at the end of an hour. In the course of the next eighteen days her condition improved remarkably; the carbon dioxide rose to 16.4 millimols per liter, and she was discharged on 0.1 gm. (1½ gr.) digitalis daily and limited fluid intake.

Final admission (one and a half months later). Following discharge the patient failed to follow directions. She was irregular in taking digitalis, drank "lots" of water and did not keep her appointment at the Out Patient Department. She became progressively more dyspneic, with increasing edema and marked orthopnea. Eight hours before admission she developed severe respiratory distress.

Physical examination showed a slightly cyanotic woman in acute dyspnea. The lung bases were dull up to the fifth interspace. Many coarse rales and wheezes were heard throughout. The breath sounds were decreased at the bases. The heart was enlarged to 4 cm. beyond the midclavicular line. A low-pitched early mid-diastolic blowing murmur was heard at the aortic region. A pericardial friction rub was audible at the base. The abdomen was negative. There was marked pitting edema of the ankles, extending to the level of the lower thorax, where only a slight pitting edema was present.

The blood pressure was 210 systolic, 110 diastolic. The temperature was 97.6°F., the pulse 120, and the respirations 30.

Examination of the blood showed a red-cell count of 3,200,000, with 5.5 gm. of hemoglobin. The white-cell count was 6400, with 60 per cent neutrophils. The urine was cloudy, with a specific gravity of 1.008 and a pH of 7.5; it gave a ++++ test for albumin and contained a trace of acetone. The sediment had 1 or 2 white cells per high-power field. The nonprotein nitrogen was 140 mg. per 100 cc., and the carbon dioxide 22.3 millimols per liter; the protein was 6.0 gm. per 100 cc., with an albumin-globulin ratio of 1.2. An electrocardiogram showed a rate of 95. The PR interval was 0.12 second. Slight left-axis deviation was present. ST_1 and ST_3 were slightly depressed, with low inversion of T_1, T_2 and T_3. Q_3 was deep, and the T wave was low in CF_4 and low and inverted in CF_5.

She was given 0.011 gm. of morphine sulfate and two transfusions of 250 cc. of whole blood each. She was also given 0.2 gm. of digitalis for two days, followed by 0.1 gm. daily, 1 gm. of ammonium chloride four times a day (for four doses), followed by 2.2 cc. of Mercupurin intravenously, and daily infusions of 25 per cent dextrose in water.

On the fourth hospital day she vomited several times. The urine continued to remain alkaline and to give a +++ or ++++ test for albumin. The basal rales diminished. The friction rub was still present, but the aortic diastolic murmur could not be heard. The blood pressure was 210 systolic, 100 diastolic. The nonprotein nitrogen was 120 mg. per 100 cc. The carbon dioxide was 20.2 millimols per liter, the chloride 93.3 milliequiv., and the blood sugar 155 mg. per 100 cc. An x-ray film of the chest showed fluid in both pleural cavities. The urinary output was 500 cc. on an intake of 1250 cc.

On the ninth hospital day pitting edema was noted extending to the neck. The pericardial friction rub was heard in the third interspace to the left of the sternum. On the eleventh hospital day 1200 cc. of clear, yellow fluid was withdrawn from the right thorax. The specific gravity of the fluid was 1.005 and it contained 180 cells per cubic millimeter, with 10 per cent red cells, 45 per cent lymphocytes, and 45 per cent monocytes. On the thirteenth hospital day she had severe, acute respiratory distress and considerable discomfort because of severe edema in the legs. Another chest tap, on the fourteenth hospital day, gave 1300 cc. of fluid from the right side and 1100 cc. from the left. The dyspnea improved temporarily but during the following week it increased. On the twenty-third hospital day Southey tubes were inserted in both ankles and thoracentesis yielded 1000 cc. from the right and 800 cc. from the left, with much improvement in the dyspnea. In the next three weeks, however, repeated chest taps were necessary. On the forty-ninth hospital day she became nauseated and began to vomit. She had considerable headache. The nausea and vomiting persisted despite atropine. The pericardial friction rub became louder; the nonprotein nitrogen was 108 mg. per 100 cc. The vital capacity was 1300 cc. She continued to get digitalis.

Her condition became progressively worse, and she died on the sixty-third hospital day.

Differential Diagnosis

Dr. Earle M. Chapman: Through an error I received the wrong clinical abstract but I shall attempt to discuss this case without having prepared it. In other words, it resolves into the type of

clinicopathological conference that Dr. Richard C. Cabot conducted years ago, when he was handed the abstract of the history on the spot.

It seems reasonable that a diagnosis of pneumonia was made following the episode of chills and fever, then pain and cough. The next attack of chest pain and dyspnea lasted for five hours. Was it substernal?

DR. BENJAMIN CASTLEMAN: It does not say.

DR. CHAPMAN: The pain was squeezing and agonizing, so I suspect that it was substernal.

Hypertension was discovered a year before entry, and the patient had edema of the ankles at that time. On physical examination advanced arteriosclerotic changes were found in the eyegrounds.

According to the laboratory notes the red-cell count was surprisingly low, and it is interesting that the specific gravity of the urine was so low (1.012). When a patient is dehydrated at entry one always looks at the first gravity, because it is of more significance than subsequent tests in the hospital, since the patient is usually given extra fluid. I assume this was an admission specimen.

DR. CASTLEMAN: The specific gravity of the urine was at first 1.012, then 1.010, 1.008 and 1.020.

DR. CHAPMAN: The record states that after eighteen days the patient's condition improved. I am not so impressed with the improvement.

The electrocardiographic changes are more than consistent with left ventricular enlargement; they suggest damage to the anterior wall of the myocardium.

The patient had a potential diabetic background. The blood sugar was slightly above normal, but not very high. She evidently was diabetic and also hypertensive. I do not see much point in giving 4 units of insulin three times a day, since this is an unnecessary burden to place on a patient. She might have taken one dose or regulated her diet.

A year before admission she developed hypertension and then began to have swelling of the ankles.

She entered the hospital with the story of being well until the sudden onset of chills and fever,[1] the diagnosis of pneumonia at the left base having been made. Pulmonary infarction is often treated as pneumonia because the signs and clinical story are often similar. Since this patient continued to have symptoms, in retrospect one has to consider infarction strongly. The second attack may have been due to more pulmonary emboli. She had changes in the circulation. Linton and his co-workers[1] in a recent publication covering 202 cases of pulmonary embolism and infarction state that 70 per cent occur in the left lower lobe, although most reports, including that of Hampton and Castleman,[2] name the right lower lobe as the favorite site. I think that is significant. I believe that this patient had an embolus and not true pneumonia.

The next question is, Was this true angina or did she have coronary occlusion with myocardial infarction of the anterior wall? The fact that it lasted five hours suggests that she had infarction. That is too long for angina.

Then she re-entered the hospital and died in uremia. I think that the persistent pneumonia, the persistent hypertension, the persistent edema and the whole clinical story — the gradual going down hill and death in uremia after a long hospital entry — favor the thing that we have been led to suspect, that is, an intercapillary glomerulosclerosis, which has been described by Kimmelstiel and Wilson[3] and which happens so often in diabetics. I believe that Dr. Castleman will show us kidneys with deposits between the capillary tufts. Edema is an outstanding clinical feature in these patients. Certainly this woman had edema, and they even used Southey tubes for it. She had pericarditis with a pericardial friction rub. As a routine interpretation we might say that the electrocardiogram showed infarction of the anterior wall with inversion of the T waves and a deep Q_3, with inversion of CF_4 and CF_5. I believe, however, that she did not have an occlusion, because the pericarditis could have produced these changes. In other words coronary occlusion with infarction in the anterior wall can be simulated in all respects by pericarditis, with or without effusion. An excellent paper on that point has just been published by Wolff,[4] who reported 5 cases seen at the Beth Israel Hospital.

The development of the blowing diastolic murmur does not disturb me. I do not believe that we have to think of aortic regurgitation, because the murmur disappeared when the patient became compensated.

I wish that Dr. Butler would comment on the blood chemical findings.

DR. ALLAN M. BUTLER: One gram of ammonium chloride daily had marked effect in producing an acidosis, the carbon dioxide dropping to 13 millimols and the chloride rising to 114 milliequiv. I think that makes sense in a patient who has marked renal damage and hence does not produce ammonia and cannot excrete sodium and chloride differentially. The next time they gave 1 gm. of ammonium chloride four times a day, but only for one day.

I suppose that this patient had marked arteriosclerosis and vascular nephritis. I wonder about the diabetes. She probably had it, but we are given no real evidence except that someone said so.

DR. CHAPMAN: She was given insulin; in other words, someone thought she had it. We do not know how limited the diet was in carbohydrate.

DR. BUTLER: There is no evidence of diabetes in the story given us, and we have to assume that it was present. On the other hand, she was not paying much attention to diet, not taking insulin and not showing much evidence of diabetes.

DR. CHAPMAN: I am interested that she put out 5 per cent of phenolsulfonephthalein dye at the end of

one hour. Before we[5] studied the fractional excretion of dye in nephritic patients, it was difficult to estimate prognosis on the basis of any single test. Our work showed that there were no survivals after one year if the dye excretion fell to 5 per cent or less in fifteen minutes, and it has proved on many occasions to have prognostic value. This case substantiates that.

DR. BUTLER: How about the weight? When they gave ammonium chloride, the serum protein increased. One wonders if that was an error on the part of the laboratory or whether the serum protein went up because of loss of edema and of weight.

DR. CHAPMAN: No weight is given on the first admission.

DR. BUTLER: If one is treating patients with cardiac failure and renal disease, an excellent way to tell what is going on is to weigh them now and then.

DR. CHAPMAN: My diagnoses are diabetes, arteriosclerosis, hypertension and intercapillary glomerulosclerosis — the type described by Kimmelstiel and Wilson. I believe that she died in kidney failure from that type of kidney disease and that she had an old pulmonary infarction at the left base. I do not believe that she had recent coronary occlusion with infarction of the anterior myocardial wall, but that these electrocardiographic changes were produced by pericarditis and effusion; possibly there was an old coronary occlusion that dated back to the first episode, two months before admission.

DR. WILLIAM BECKMAN: The patient had lost 20 pounds. On entry she weighed 139 pounds, and two weeks later 119.

DR. BUTLER: We can assume then that the ammonium chloride had a diuretic and a dehydrating effect, with the increase in serum protein that Dr. Chapman noted.

DR. BECKMAN: During the first admission she definitely improved clinically so far as decompensation was concerned. When I went over her the first time I thought of intercapillary glomerulosclerosis, but finally ruled it out because she did not have the retinopathy typical of diabetes. The retinal changes were characteristic of arteriosclerosis. So I decided that she had arteriosclerotic kidneys and hypertension.

DR. BUTLER: Can you tell us whether she really did have diabetes?

DR. BECKMAN: We were not certain, since we merely did the routine tests. She continued to excrete a little sugar each day. She never had a blood sugar higher than 155 mg. per 100 cc., and she was given insulin on the theory that all diabetic patients should receive insulin for three days.

DR. CHAPMAN: Was a glucose-tolerance test done? I am impressed by the number of patients in the Out Patient Department on whom a diagnosis of diabetes is made on a single test showing sugar in the urine. It seems that glucose-tolerance tests are not done often enough to establish a definite diagnosis.

DR. BUTLER: In many of the patients you are speaking about there is no doubt about the diabetes. A typical history, a high blood sugar and sugar in the urine usually establish the diagnosis of diabetes. Thus there is no need of doing a glucose-tolerance test. In the doubtful case it is reasonable to ask for such a test.

DR. CHAPMAN: I agree.

CLINICAL DIAGNOSES

Chronic vascular nephritis, with uremia.
Hypertensive heart disease.
Congestive failure.

DR. CHAPMAN'S DIAGNOSES

Diabetes.
Arteriosclerosis.
Hypertension.
Intercapillary glomerulosclerosis.
Uremia.
Pericardial effusion.
Pulmonary infarction, left base?
Coronary occlusion (two months old)?

ANATOMICAL DIAGNOSES

Intercapillary glomerulosclerosis, marked.
(Uremia.)
Diabetes mellitus.
Myocardial infarction, recent and old.
Coronary sclerosis with old occlusion.
Cardiac hypertrophy, hypertensive type.
Hydrothorax, bilateral.
Bronchopneumonia, terminal.
Pericarditis, healing.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The autopsy on this patient showed a hypertrophied heart weighing 450 gm. The coronary arteries were markedly sclerotic, and in the descending branch of the left we found an old occlusion, with a corresponding healed infarct in the myocardium. In addition there were scattered through the left ventricular wall yellow and pinkish-red areas that looked like the lesions of acute infarction. Microscopic examination showed acute myocardial infarction, although we were unable to find a recent thrombosis. This infarction showed polymorphonuclear infiltration and necrosis of muscle fibers, so that I do not believe that there is any question about the diagnosis. Also, the involved areas were in the endocardial side of the myocardium rather than in the pericardial side, so that they were not due to an extension from the

pericarditis, which at the time of autopsy was in the healing stage. There has been a lot of discussion about the appearance of the myocardium in patients with renal failure. Most people believe that there is no definite myocarditis due to renal failure, although Gouley[6] has stated that there is a characteristic myocardial degeneration.

DR. CHAPMAN: How extensive was the recent infarction?

DR. CASTLEMAN: It was very extensive and certainly not more than a week or two old.

DR. CHAPMAN: Then it could not explain the changes in the electrocardiogram taken sixty days before.

DR. CASTLEMAN: No.

DR. CHAPMAN: They might well have been explained on the basis of the acute pericarditis that she had had at the time.

DR. CASTLEMAN: Yes.

The kidneys weighed 250 gm. and showed numerous scars throughout. Microscopically we found the condition that Dr. Chapman predicted. Most of the glomeruli showed extensive hyalinization in such a fashion as to give the tufts a lobulated appearance, the sclerosis being predominantly intercapillary. Many of the peripheral capillaries were patent. This is the characteristic picture described by Kimmelstiel and Wilson.[3]

DR. BUTLER: How about the small arterioles?

DR. CASTLEMAN: There was a goodly amount of vascular nephritis as well.

The only remnant of the old process in the left lower lung was a fibrous diaphragmatic pleuritis. Unfortunately, sections through the underlying parenchyma were not taken for microscopic examination, so that we do not know whether the lesion was due to an infarct or pneumonia.

REFERENCES

1. Allen, A. W., Linton, R. R., and Donaldson, G. A. Thrombosis and embolism: review of 202 patients treated by femoral vein interruption. *Ann. Surg.* **118**:728-740, 1943.
2. Hampton, A. O., and Castleman, B. Correlation of postmortem chest teleroentgenograms with autopsy findings, with special reference to pulmonary embolism and infarction. *Am. J. Roentgenol.* **43**: 305-326, 1940.
3. Kimmelstiel, P., and Wilson, C. Intercapillary lesions in glomeruli of kidney. *Am. J. Path.* **12**:83-98, 1936.
4. Wolff, L. Acute pericarditis simulating myocardial infarction. *New Eng. J. Med.* **230**:422-425, 1944.
5. Chapman, E. M., and Halsted, J. A. Fractional phenolsulfonephthalein test in Bright's disease. *Am. J. M. Sc.* **186**:223-232, 1933.
6. Gouley, B. A. Myocardial degeneration associated with uremia in advanced hypertensive disease and chronic glomerular nephritis. *Am. J. M. Sc.* **200**:39-49, 1940.

CASE 30352

PRESENTATION OF CASE

A twelve-year-old schoolboy entered the hospital complaining of pain in the right hip.

The patient had been well until three months before admission, when he slipped and fell on his right hip. He developed pain and limitation of motion which became progressively worse two to three weeks before admission.

Physical examination showed a well-developed, well-nourished boy in no acute distress. The heart, lungs and abdomen were negative. There was tenderness over the lateral aspect of the right hip, and marked limitation of motion due to spasm and pain.

The blood pressure was 125 systolic, 80 diastolic The temperature was 100°F., the pulse 85, and the respirations 25.

Examination of the blood showed a white-cell count of 8600, with 71 per cent neutrophils, 22 per cent lymphocytes, 4 per cent monocytes, 2 per cent basophils and 1 per cent eosinophils. The hemoglobin was 12.8 gm. per 100 cc. The urine was normal. A blood Hinton test was negative.

Anteroposterior stereoscopic films of the pelvis showed destruction of the right ilium along the entire region of the right sacroiliac joint (Fig. 1). The process appeared to extend well into the ilium and to involve the medial half of the iliac crest. The joint itself and the sacral side of the joint did not appear to be involved. There was a soft-tissue mass overlying the right ilium that measured 9 cm. in diameter. X-ray films of the spine showed moderate scoliosis toward the right, which might have been due to splinting and muscle spasm. The kidney shadows were of average size and shape and in the usual position. There were no other areas of destruction in the pelvis, spine or the thoracic cage. Roentgenogram of the chest was negative.

Following admission, the patient seemed to be fairly comfortable so long as he was confined to bed. He continued to run a low-grade fever. Other white-cell counts were 13,900 and 19,200.

On the second hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. CHANNING C. SIMMONS: There are some data that are not given in the protocol that might be of value in making a diagnosis. The blood chemical findings, for instance, would be of value in ruling out certain bone tumors, and a tuberculin test would help in ruling out tuberculosis of the sacroiliac joint. No mention is made of lymph-node involvement.

DR. BENJAMIN CASTLEMAN: The serum calcium was 9.4 mg. per 100 cc., the phosphorus 4.3 mg., and the phosphatase 1.7 Bodansky units. A tuberculin test was not done.

DR. SIMMONS: In other words the phosphatase was on the low side of normal. The serum protein would be of value in ruling out plasma-cell myeloma, which I doubt that this boy had.

Are the x-ray films available?

DR. LAURENCE L. ROBBINS: These films show a destructive process involving the ilium, with apparent extension up to but not involving the

sacroiliac joint. The soft-tissue mass definitely extends into the pelvis.

DR. SIMMONS: What is the shadow at the hilus of the right lung?

DR. ROBBINS: It is possibly due to enlarged hilar nodes, but I could not be positive without fluoroscoping the patient.

DR. SIMMONS: The physical examination says that the abdomen was negative, and makes no mention of what, in the film, is obviously a shadow occupying the greater part of the pelvis.

FIGURE 1. *Roentgenogram of Pelvis.*

In all cases of bone disease there are three groups of diseases to consider: first, an inflammatory condition, such as tuberculosis, osteomyelitis or syphilis; second, a systemic disease such as parathyroid or Paget's disease; and, third, some form of bone tumor, benign or malignant and either primary or secondary. I frankly cannot make a definite diagnosis on this boy, but on the basis of the facts given in the protocol and the x-ray findings I have to consider these three conditions.

Of the inflammatory conditions, because of the low-grade temperature, the history of trauma and the bone destruction, he might have had an osteomyelitis. I am not particularly concerned with tuberculosis, since it usually arises around the joint rather than in the body of the bone. I can rule out syphilis on account of the negative Hinton test.

I do not know of any systemic disease that gives an appearance of tumor, with bone destruction limited to one bone, associated with an elevated temperature and white-cell count.

Then there are the tumors. It might have been metastatic from either a tumor of the kidney or a neuroblastoma of the mediastinum or suprarenal gland. It might have been a primary bone tumor. An osteogenic sarcoma in this situation is unusual. A Ewing's sarcoma is quite common and often results in a somewhat elevated white-cell count and a low-grade fever. There is one other tumor that can simulate in bone practically every condition that I know, and that is a malignant lymphoma. A lymphoma can cause bone destruction and give a picture like that of osteomyelitis.

I frankly cannot make a diagnosis in this case. I believe that it was probably an osteomyelitis but cannot rule out a lymphoma or a Ewing's sarcoma. I should want a biopsy.

DR. ALLAN M. BUTLER: You would want a biopsy before considering therapy?

DR. SIMMONS: Very definitely. There is no use in giving therapy unless one knows what one is

treating. If it is malignant disease, one can give a sufficiently heavy treatment; whereas if it is an inflammatory condition, this method of treatment should not be used. The radiologists are loath to treat any condition by x-ray unless a biopsy has established a diagnosis.

Clinical Diagnosis

Osteomyelitis of ilium.

Dr. Simmons's Diagnosis

Osteomyelitis of ilium?
Malignant lymphoma?
Ewing's sarcoma of ilium?

Anatomical Diagnosis

Ewing's sarcoma of ilium.

Pathological Discussion

Dr. Castleman: Apparently this mass must have been felt, as Dr. Simmons suggested, because they were able to puncture it with a needle and to aspirate a good-sized biopsy specimen. Microscopically the lesion proved to be a Ewing's sarcoma.

Preoperatively it was believed to be an osteomyelitis because of the sepsis, the high white-cell count and the fever. Following the biopsy the child was given a course of x-ray treatment and sent home. I recently heard from Dr. G. W. Van Gorder, who followed the patient, that within six weeks the child died of diffuse metastases to the lungs. I suppose that the shadow that Dr. Simmons pointed out in the chest film could have been a metastasis. Is that so, Dr. Robbins?

Dr. Robbins: Probably.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal

Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Richard M. Smith, M.D., *Chairman*
James P. O'Hare, M.D. William B. Breed, M.D.
Conrad Wesselhoeft, M.D. Oliver Cope, M.D.

Official Organ of
The Massachusetts Medical Society
and
The New Hampshire Medical Society

Editorial Board
Joseph Garland, M.D. A. Warren Stearns, M.D.
Shields Warren, M.D. Dwight O'Hara, M.D.
C. Guy Lane, M.D. Chester S. Keefer, M.D.
William A. Rogers, M.D. Howard M. Clute, M.D.
Stephen Rushmore, M.D. Fletcher H. Colby, M.D.
Henry R. Viets, M.D. Robert L. Goodale, M.D.
Robert M. Green, M.D. Chester M. Jones, M.D.
Charles C. Lund, M.D. Harvey R. Morrison, M.D.

Associate Editors
Thomas H. Lanman, M.D. Donald Munro, M.D.
Henry Jackson, Jr., M.D.

Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year (Boston funds); $8.52 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

POSTWAR PLANNING

Of the approximately 55,000 medical officers now in military service, 3000 have been asked to express themselves concerning postwar medical training. Of these, 927 had replied up to June 1, and the answers to the questionnaire are reported in the June 24 issue of the *Journal of the American Medical Association*. If this sample is representative of the whole group, — and there is no reason to think that it is not, — a large order in postwar medical education is about to be placed.

Divided according to the dates of licensure, which are roughly parallel to the dates of graduation from the medical school and therefore to age groups, the 927 officers fell into the following groups: Group 1, 400 (43 per cent); Group 2, 323 (35 per cent); Group 3, 168 (18 per cent); and Group 4, 36 (4 per cent). Group 1 consisted of those licensed from 1937 to 1943; Group 2, those licensed from 1930 to 1936; Group 3, those licensed from 1920 to 1929; and Group 4, those licensed before 1920.

Of these Group 1 is the largest, and those contained in it are the youngest and the most in need of postwar education. Many men in this group had their periods of medical training shortened and hurried by the war, so that it is not surprising that 90 per cent of them state that they will want between one and two years of additional training. If the ratio of 400:927 holds for the 55,000 officers, the total number of Group 1 men — those who graduated subsequent to 1936 — will approximate 24,000.

Already about 300 of these 400 officers, or about 18,000 of the total, have stated that they wish to specialize, which means that residencies will be in demand. A previous survey has shown that there are in this country about 8000 residencies, and it was suggested that these facilities might be expanded to accommodate 12,000 in the immediate postwar period.* The necessity for a gradual demobilization is apparent if all the officers wishing training are to be accommodated without preliminary waiting periods, for there will be a greater load than the teaching hospitals of the country can possibly absorb in any one-year or two-year period. The replies of these young medical officers also indicate that rural communities will continue to find it difficult to attract doctors. Only 6 per cent expressed a preference for a rural location, the majority preferring a community of between 25,000 and 250,000 population.

To what extent this thinking on the part of the small sample analyzed represents the whole and to what extent the thinking of the whole may undergo future changes remain to be decided. For the present one can only visualize a postgraduate medical education program of unprecedented proportions — something that will have to bring into use a larger proportion of clinical material than has ever before been utilized.

*Medicine and the war: postwar graduate medical education. A preliminary report by the Council on Medical Education and Hospitals. *J. A. M. A.* **124**:40, 1944.

WHY IS A JAP?

THE magazine *Time*, although nor ordinarily used as a journal of scientific reference, contains in its August 7 issue two items of more than ordinary interest regarding the Japanese behavior pattern. The first is an account by Robert Sherrod, *Time* correspondent, of the mass suicides on Saipan. The second is the news story of an analysis of Japanese character by Geoffrey Gorer, anthropological investigator in the Institute of Human Relations at Yale University.

The western world has become thoroughly accustomed to bizarre stories of the picturesque Japanese hara-kiri ceremony, considering the idea of it as something remote from our own usual mental processes. We know of how, in times of peace, the sober-minded Japs were wont to toss themselves into the craters of hyperactive volcanoes; from our marines in the South Pacific and our soldiers on Attu and Kiska we have more recently heard of the wholesale and apparently purposeless self-destruction practiced by the soldiers of the Emperor when faced with final defeat. This has frequently been not a matter of fighting to the last ditch. Soldiers have impaled themselves on the bayonets of the enemy or have fallen on their own swords or blown themselves to bits with hand grenades when they still had ammunition with which to continue fighting.

On Saipan, for the first time, our forces gained possession of territory with a considerable settled Japanese population, and so prevalent became suicide among the civilians of both sexes and all ages, according to Sherrod, that the question is raised whether the whole Japanese race will choose death to surrender.

Gorer, in his study of Japanese psychology, and with perhaps more than a slight leaning toward the Freudian concept, places great emphasis on the severe toilet training of the Japanese infant. Beginning at the age of four months the baby is held over the balcony or road at frequent intervals and for obvious reasons not at all suggestive of the Capulet and Montague affair. Lapses in conduct are punished with great severity. At six months he is taught to bow respectfully (although the polite hissing through the teeth is waived), and before the end of the first year "he is forced to learn to sit stiffly on his haunches."

The male is taught that he belongs to the dominant sex, but even so his life is bound rigidly by convention. To overstep a rule of behavior brings ridicule without mercy from friend and foe, and so we find developed the repressions that have their outlet in cruelty and arrogance toward supposedly weaker individuals and races, and the dread of loss of face, which makes life intolerable in an environment that has become hostile.

We thus see in the rigid training of the Japanese both his strength and his weakness. It drives him on through hardship and danger with the endurance of a fanatic, but when it fails to achieve its goal he is finished. If we accept the thesis, however, that the modern Japanese is the product of an unfortunate environment and not a completely strange and different biologic mishap, then there is some hope of adapting future generations of the race to existence in a world that recognizes the rights of others.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

BROWN — Edison W. Brown, M.D., of Revere, died August 18. He was in his sixty-ninth year.

Dr. Brown received his degree from Tufts College Medical School in 1905. He was a member of the American Medical Association and the New England Obstetrical Society and a staff member of the Chelsea Memorial Hospital, Whidden Memorial Hospital, Everett, and Winthrop Community Hospital.

His widow, his mother and two sisters survive.

COBB — Farrar Cobb, M.D., of Hyannis, died May 28. He was in his seventy-eighth year.

Dr. Cobb received his degree from Harvard Medical School in 1893. He served on the staff of the Massachusetts General Hospital and as superintendent of the Massachusetts Eye and Ear Infirmary. He was chief surgeon from 1930 to 1935 and on the consulting staff from 1930 to 1944 of the Cape Cod Hospital. He was a member of the American Medical Association and the New England Surgical Society and fellow of the American College of Surgeons.

DUNN — Lt. Raymond A. Dunn, of Worcester, was killed when the destroyer on which he was medical officer was sunk by enemy fire in the invasion of Sicily. He was in his thirty-fourth year.

Dr. Dunn received his degree from Tufts College Medical School in 1935. He was an obstetrician on the staff of St. Vincent Hospital. He was a member of the American Medical Association and the Worcester District Medical Society.

His parents, a sister and a niece survive.

BREED — William B. Breed, M.D., of Newton, died August 21. He was in his fifty-second year.

Dr. Breed received his degree from Harvard Medical School in 1920. At the time of his death he was a member of the Committee on Publications and a councilor of the Massachusetts Medical Society, and chairman of its War Participation Committee. He was associate editor of the *Journal* from 1923 to 1937, and a member of the editorial staff from 1937 to 1942. He was a staff member at the Massachusetts General Hospital and an associate in medicine at Harvard Medical School. He was also a member of the American Medical Association, the American College of Physicians, the New England Heart Association and the American Clinical and Climatological Society.

His widow and three children survive.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATH

SARGENT — Frank H. Sargent, M.D., of Pittsfield, New Hampshire, died July 11. He was in his eighty-third year.

Dr. Sargent received his degree from Dartmouth Medical School in 1889. He was a member of the American Medical Association, the New Hampshire Medical Society, and the Merrimack County Medical Society.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN

Clinic	Date	Clinic Consultant
Haverhill	September 6	William T. Green
Lowell	September 8*	Albert H. Brewster
Salem	September 11*	Paul W. Hugenberger
Brockton	September 14	George W. Van Gorder
Worcester	September 15	John W. O'Meara
Pittsfield	September 18	Frank A. Slowick
Springfield	September 20	Garry deN. Hough, Jr.
Fall River	September 25	Eugene A. McCarthy
Hyannis	September 26	Paul L. Norton

*Date changed.

CORRESPONDENCE

ANTERIOR POLIOMYELITIS

To the Editor: I have been much interested lately in reading in *Life* the account of the work being done by medical men in North Carolina during the existing epidemic of infantile paralysis.

The statement therein to the effect that these doctors are examining flies in the search for probable means of transmission brings back to my mind very distinctly my experience with that disease in this city some thirty years ago. At that particular time, since I was chairman of the Board of Health, and in contact with the official representative of the State Board of Health, as well as an epidemiologist, I was in a position to see most of the cases of that disease — not only my own patients, but many others; and I was much impressed at the time in observing on the bodies of many of the victims small discolored, concrete areas, which clearly had the appearance of being insect bites.

In my report to the State Board of Health I called attention to this fact. Nothing came of it, however; but I believed at the time, and have believed ever since, that there was to be sought the possible cause of the invasion.

Furthermore, it appeared that shortly before the first cases were reported, — which happened to be my own, — there were three cases in one locality where a number of pigs kept in the vicinity all died within a short time; and it was said that, before they died, each one was a victim of paralysis. In my opinion this was due to some insect with a very limited radius of activity, as the disease was not general, but confined to certain territories, where the afflicted persons were adjacent to one another.

I hope this study in North Carolina will bring forth something worth while.

William H. Keleher, M.D.
48 Pleasant Street
Woburn, Massachusetts

NOTICES

ANNOUNCEMENT

Dr. Alice Nauen announces the removal of her office from 121 Sutherland Road, Brookline, to 41 Concord Avenue, Cambridge 38.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, September 7, in the classroom of the Nurses' Residence at 7:15 p.m. Dr. Louis K. Diamond will speak on the subject "The Rh Factor." Dr. Hildur Gibson will be chairman.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, September 7

Thursday, September 7

7:15 p.m. The Rh Factor. Dr. Louis K. Diamond. New England Hospital for Women and Children.

Saturday, September 9

*10:00–11:30 a.m. Medical staff rounds. Peter Bent Brigham Hospital.

Monday, September 11

International Association of Industrial Accident Boards. Hotel Statler, Boston.

*12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

Tuesday, September 12

International Association of Industrial Accident Boards. Hotel Statler, Boston.

*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

Wednesday, September 13

International Association of Industrial Accident Boards. Hotel Statler, Boston.

*12:00 m. Clinicopathological conference. Children's Hospital.

*Open to the medical profession.

September 6–9. American Congress of Physical Therapy. Page 248, issue of August 10.

October 2–7. Seminar in Legal Medicine, Harvard Medical School. Page 110, issue of July 20.

October 3–5. American Public Health Association. Page ix, issue of March 30.

October 4. Medicolegal conference, Mallory Institute of Pathology, Boston. Page 110, issue of July 20.

October 9–20. 1944 Graduate Fortnight of the New York Academy of Medicine. Page xvii, issue of July 27.

October 16–November 3. Third Postgraduate Course in Industrial Medicine.

October 30. New York Institute of Clinical Oral Pathology, New York Academy of Medicine. Page 110, issue of July 20.

November 2–4. Association of Military Surgeons. Page xiii, issue of August 17.